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TIBULAR TESTS IN INTRACRANIAL SURGERY

(FIRST PAPER)

THE IMPORTANCE (1) OF THE ABSENCE OF REACTIBILITY OF THE VERTICAL CANALS OF BOTH EARS TO STIMULATION BY THE COLD CALORIC IN THE DIAGNOSIS OF INCREASED INTRACRANIAL PRESSURE OF THE POSTERIOR FOSSA, AND (2) OF THE REDUCTION OF THE DURATION OF THE NYSTAGMUS FROM TURNING IN DIFFUSE LESIONS OF THE CEREBROSPINAL SYSTEM PATHWAYS OVER THE CEREBELLAR CORTEX (AS IN PROTECTIVE MENINGITIS AND LINEAR FRACTURES OF THE SKULL OF THE POSTERIOR FOSSA).

BY

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NEWARK, N. J.

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By WELLS P. EAGLETON, M. D., Newark, N. J.

SYNOPSIS.

I. Technic:

A department of Neuro-otology under the direction of an otologist, but largely operated by a trained technician, is advised in all hospitals.

II. Auditory Apparatus:

(a) During the evolution of life the auditory apparatus was a comparatively late development: it is a very highly specialized function.

(b) The accepted hearing tests are largely valueless for differential diagnostic purposes as far as the central portion of the auditory apparatus is concerned.

III. Vestibular Apparatus:

(1) A vestibular apparatus for orientation is well developed in very low forms of invertebrate life; it is one of the earliest of the special senses. Because of its early development its function has become largely an inherent property of the nervous tissue. It is doubtful whether any one special central tract is indispensable to the performance of orientation. Consequently, injury to or destruction of a vestibular tract or pathway is quickly and at times completely compensated for.

*Presented in parts before the American Laryngological, Rhinological and Otological Society at its annual meeting in Atlantic City, June 1, 1921; the American Clinical College of Surgeons in Philadelphia, October, 1921; the New England Oto-Laryngological Society, Boston, March 30, 1922; and the American Laryngological, Rhinological and Otological Society, Washington, May 6, 1922.

Clinical Application:

Because of these biological facts a fundamental mistake has been made in trying to formulate rules only applicable when dealing with highly specialized tracts such as are present in the speech mechanism:

(1) Because of the low and primarily reflex (automatic) activity of both the vestibular mechanism within the cerebellum, compensation is easily established; consequently, gross surgical lesions affecting the central vestibular apparatus, if of slow growth, may produce either very transient spontaneous symptoms, or, in chronic lesions, no symptoms whatever.

(2) For the same biological reason, in interpreting the findings of the vestibular tests, the presence or absence of the following factors should always be considered, as they modify the vestibular manifestations:

(a) Is the lesion microscopic or diffuse?

(b) Has compensation had time to be established, and, consequently, is the lesión acute or chronic?

(c) Is increased intracranial pressure present?

(3) In microscopic lesions acutely destructive of a so-called "vestibular pathway" not associated with increased intracranial pressure, exact localization by means of the vestibular manifestations alone frequently is possible; as in minute hemorrhages, disseminated sclerosis and syringomyelitis.

(4) The vestibular manifestations of increased intracranial pressure, viz., the reduction of the reactibility of the vertical canals of both ears to the cold caloric (the reduction of the opposite side in the author's opinion, being the greater) is of the greatest clinical value.

(5) In surgical lesions, because of the rapid and more or less complete establishment of compensation and the overshadowing vestibular manifestations of increased intracranial pressure, localization of lesions by an examination of the vestibular apparatus alone is difficult, and generally impossible.

(6) Certain localized cerebellar surgical lesions, such as acoustic tumors, frequently can be diagnosed by the vestibular tests alone. The vestibular manifestations in such cases are a combination (1) of the effect of direct pressure on the vestibular tracts between the labyrinth and the brain stem as shown by the homolateral deafness, the absence of reactibility of all the homolateral canals, the spontaneous falling, and (2) the effect of the increased intracranial pressure of the posterior fossa in abolishing the reactibility of the vertical canal of the opposite side.

(7) In surgical intracerebellar lesions, such as gliomas or diffuse suppurative lesions (abscesses), localizing evidence may or may not be obtainable from the vestibular reactions; but the findings frequently are so transient and changeable, and as yet so imperfectly understood, that localizing surgical diagnosis can rarely be fundamentally based upon the findings of the vestibular tests alone.

(8) However, at any moment, due to an invasion of an intracerebral portion of the vestibular apparatus, spontaneous or induced, vestibular manifestations may appear, of localizing value—spontaneous vertical nystagmus, lateral deviation of the eyes—all of which usually rapidly disappear because of compensation.

(9) In exceptional cases, the spontaneous vestibular manifestations continue for indefinite causes probably due to invasion of, but without destruction of, the vestibular nuclei in the brain stem.

Clinical Applications:

(10) The immediate reduction of the duration of the nystagmus obtainable from stimulating the horizontal semi-circular canals by turning, is of distinct surgical and medico-legal value in diagnosing certain diffuse lesions of the posterior fossa. In the author's experience, this occurs in those lesions which are associated with a blockage of the cerebrospinal fluid pathways by exudate, such as in protective meningitis or by blood in fracture of the skull, chiefly of the posterior fossa.

Conclusions:

(11) These two vestibular manifestations, viz., of increased intracranial pressure of the posterior fossa, and of blockage of the cerebrospinal fluid system pathways of the posterior fossa, should always be considered in all attempts at localization of surgical lesions of the posterior fossa. In the author's experience they are the two chief intracranial vestibular manifestations constantly present so as to be of consistent practical diagnostic value in cerebral surgery.

There is clinical evidence that sudden invasion of the frontal lobe causes spontaneous past-pointing. This rapidly disappears. There is also clinical evidence that too sudden invasion of the temporal lobe causes loss of quick component in induced nystagmus by application of the cold caloric applied to the side of the lesion. This also rapidly disappears.

There is clinical evidence that sudden involvement of the cerebellum will cause nystagmus or a lateral deviation of the eyes. Both of these also rapidly disappear although in certain lesions, such as tumor and irreparable damage to the cerebellar nuclei the nystagmus persists.

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VESTIBULAR AND AUDITORY TESTS IN INTRACANIAL SURGERY.

I. Technic of Otological Examinations in General.

At present the routine aural examinations in our large hospitals, if conducted at all, consist of an inspection of the drum membrane and a test to ascertain if the patient hears with each ear separately. Yet, in every general hospital there is constantly a mass of material which, if a systematic effort were made to correlate it, would throw light on this complicated subject.

In my opinion, all cases that enter the surgical wards for cranial injuries should have during treatment or at least before leaving the hospital, a routine otological examination by the functional tests. The same should apply to all cases in the medical wards which complain of vertigo, tinnitus or deafness; while, of course, no neurological examination should be regarded as complete without containing a full record of the vestibular reactions, both spontaneous and induced.

To make this possible there should be established in all general hospitals a department in charge of an otologist, although in my opinion the techical part of the work should not be required of him personally. All routine technical detail should be delegated to a male or female technician of intelligence. It is asking too much of the otologist to require him to devote the large amount of time necessary for the tests, with—in the majority of cases—negative findings. It would be as reasonable to ask the attending physician or surgeon to perform the routine urine examination.

The technician should be instructed by the otologist how to conduct the tuning fork tests; how to measure the duration of the bone conductions and to perform calorics on the vertical and horizontal canals. His report should be turned over to the otologist, and from it the otologist should make his diagnosis, verifying all abnormal findings. A few hours devoted to the instruction of such a technician in the use of the turning chair and the cold caloric—at first on a series of normal cases, followed later by abnormal ones—would soon make this possible. For the chief requisite is not medical knowledge, but keen powers of observation, associated with patience in execution, truthfulness in recording, and enthusiam for the work. Until this division of the technical work is generally established, we shall make little progress in the vestibular tests.

The above is put forward, not as a suggestion, but as a special plea, because work so conducted has been admirably successful in the two instances in which I have had authority to enforce it.

II. Audition Tests in Cerebral Localization.

Evolutionary Facts of Clinical Value:

Audition is one of the late developmental additions in the evolutionary ascent of life. It is by means of the auditory function that speech was evolved, and from speech have come all the still higher psychic attributes possessed by man. Conse-

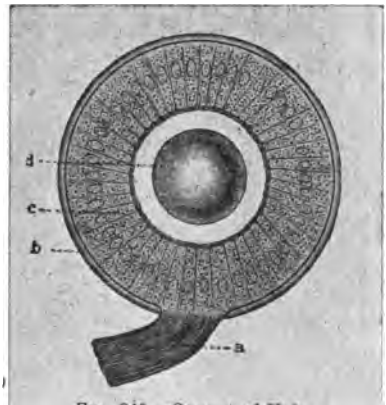


Fig. 240.—Otocyst of Unio.
a, acoustic nerve; *b*, capsule of the otocyst in which it ramifies; *c*, vibratile epithelium; *d*, otolith.

Figure 1. Vestibular Apparatus In Bivalve (River Mussel) Showing Complete Vestibular But No Auditory Apparatus.
From Morat, J. P.: Physiology of the Nervous System. Keener, Chicago, 1906.

quently, the auditory apparatus, both central and peripheral, is highly specialized—higher even than the visual apparatus. In man one of the evidences of this is that, although there is a hearing centre situated in both hemispheres, the auditory apparatus is the only one of the special senses, outside of speech, which in certain parts *still has a tendency* to a cortical centre localized in but one cerebral hemisphere—the left; for while destruction of the hearing centre in the horizontal gyrus (Hershel's gyrus) of one temporosphenoidal lobe does not cause deafness, in certain cases injury to this gyrus on the left side will occasion word deafness.

All the other senses have in the evolution of life become represented equally in the brain's cortex of both hemispheres.

An auditory apparatus is only present in the comparatively higher forms of life, in contradistinction to the vestibular apparatus which is well developed in very low types. While an otolite (with neuro-epithelial cells, the primitive part of the vestibular apparatus) is present in a very low form of invertebrate (in one of the low sub-divisions of the coelenterata), and the primitive medusa (jelly fish) has a well developed vestibular apparatus ("an otocystic formation with ciliated cells connected by nerves to a nerve ganglion, and this, in turn, with muscles"*), there is no evidence of a hearing apparatus in the invertebrate kingdom. It is only among the vertebrates, in which the vestibular apparatus is fully developed, that a hearing apparatus first appears. From these rudimentary beginnings it is gradually developed up to the mammals and becomes complete in man. Fish have no cochlea, although possessing a well developed vestibular apparatus with a utricle and three semicircular canals. Snakes have a rudimentary cochlea (reduced to a quarter of a turn). "In birds the cochlea generally remains rudimentary, but the semicircular canals are highly developed."*

This comparatively late acquisition by animal life of this highly specialized sense, audition, explains the clinical facts:

(1) that on account of its high specialization slight injuries are apt to destroy it totally;

(2) that injuries to the apparatus are slowly or never compensated for;

(3) that it is also because of its high specialization that we have as yet no proper diagnostic means of *localizing central lesions of the auditory apparatus* outside of the clinical appearance of word deafness.

We must realize that hearing is not only a highly specialized function, but it is an intellectual—psychic—function above the reflex-subconscious sphere, in which the vestibular function fundamentally resides. To hear and interpret, we must *listen*.

Audition, however, has an intimate association with the subconscious.

A cat, after the removal of the cerebral hemispheres, striata and thalami, shows orientated movements of the pinna and neck in response to sounds, such as a bark, whistling, et cetera.

*Morat, J. P.: Physiology of the Nervous System; English Edition, 1906, p. 601.

A protective reaction by the auditory motor is the jump reflex—showing how low down in the subconsciousness is the association between the reaction to sounds and orientation, the chief end organ of the latter being the vestibular apparatus.*1 But the decerebrated cat does not hear in the higher sense in which the patient values hearing, for the purpose of intercourse with his fellows.

To hear speech we must listen, and this necessitates a motor and an intellectual effort. If we do not listen, in church, for instance, the speaker's voice soon fades away as well as the text about which he is discoursing.*2

Tests for Localizing Lesions of the Auditory Apparatus.

Although the violin for a musical instrument is the nearest approach to the human voice, in reality it bears no direct relation to it; in fact, no musical instrument can be adapted to act as a standard for speech, for the auditory apparatus of man, both peripheral and central, is especially specialized to perceive and interpret the human voice. From the viewpoint of the patient and from a proper diagnostic standpoint, it matters little how well or how poorly musical tones are heard if the modulations of the human voice are not correctly perceived.

But by some mischance the tuning fork, which furnishes a standard of *musical tones,* was adopted to differentiate the extent and position of lesions of the auditory apparatus.

It will require a much more delicate apparatus, based on a totally different principle, before we shall be able to diagnose the exact location of the lesions in the central portion of the auditory apparatus; for speech is made up of considerably more than a narrow range of musical notes.

Of the large number of musical instruments that have been put on the market as aids to *differential diagnosis of the site of the lesion, whether peripheral or central,* I should say (from a long personal experience with the following—Hartman's Set of Tuning Forks, Edelman's Forks, Gradenigo's Visual Forks, Koenig's rods, Politzer and Hays' Acumeters, and Galton's Whistle) that they are all *largely overrated.* Whatever practical information may be useful in differentiation is obtainable from one fork of long duration, excepting possibly in the case of partially deaf mutes, with whom my experience is so limited that I am not qualified to speak.

*1—Sherrington, C. S. and Forbes, A.: American Journal of Physiology, 1914, Vol. XXV, p. 367

*2—"The most satisfactory theory is that in the act of listening the resistance at the various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrites, which brings those of contiguous neurones into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the retraction of the dendrites." Hurst, Arthur F.: "Psychology of the Special Senses—Hearing, Listening, and Hysterical Deafness." The Lancet, August 7, 1920, p. 285.

On account of the great difference in the personal equation between different patients and observers, it is necessary that a tuning fork of over 200 d. v. per sec., be used uniformly to obtain even the meagre diagnostic information that the tuning fork may furnish.

III. The Practical Diagnostic Value—General and Localizing of the Vestibular Reactions in Cerebral Surgery.*

Present State of Knowledge:

The vestibular reactions of the end-organ—labyrinth—involvement are now fairly well understood, although many of the views of the physiology and pathology of the labyrinth itself are entirely hypothetical and are constantly being modified.

The anatomical and physiological knowledge of the cerebral portion of the vestibular apparatus is, however, still very imperfect.

Following the initial and enthusiastic acceptance of the localizing assistance of the vestibular tests in intracranial lesions, their practical value is to-day being seriously questioned. We must frankly admit that the subject is still in considerable confusion, and many of the findings are as yet unexplainable.

Disastrous Results of Hypothetical Teaching, not Based on Clinical or Experimenting Experience:

An injury has been done and a proper study of the tests retarded by the publication as facts of what in reality are but hypothetical deductions. Undue emphasis has been placed upon selected and isolated cases in which microscopic lesions of the cerebellar peduncles and of the posterior longitudinal bundle have been successfully diagnosed.

The positiveness of the statements created the impression that the tests, if properly performed and interpreted (from the viewpoint of adequate anatomical and physiological knowledge of the nervous system) are so accurate that a diagnosis of the localization of lesions of the medulla, pons, and parts of the cerebellum may be uniformly made (although anatomically a large part of these "tracts" still are problematical); the inference, of course, being that when such a diagnosis is not forthcoming the fault lies with the observer and not with the tests themselves.

There is no doubt that in very exceptional cases in microscopic lesions, such localizing diagnosis is possible; but the publication of a few isolated, brilliant diagnoses has discouraged the independent worker, because, failing to obtain localizing assistance

*Read in part before the American Otological, Rhinological and Laryngological Society at its Annual Meeting at Atlantic City, June 1, 1921.

in the average *surgical* case, he naturally has regarded the fault as with himself—in his lack of ability to interpret the findings correctly. The effect has been to intimidate the otologists, make skeptical the neurologists and neuro-surgeons, and, in consequence, to discourage a continuation of the work.

Fallacy of Attempting to Apply Theroretic Physiological Deduction to Surgical Lesions:

In those books in which few or no references to individual work are made, the brain is regarded as an inert body—a sort of complicated system of telephone wires, through which it is possible to prevent such and such a transmission "by interference" with such and such a tract without disturbing the other tracts; much as a lineman "cuts out" a wire.

It appeared so logical that interference with a pathway should cause loss of this or that part of a reaction that for a long time (in common with the rest of the profession) I failed to perceive the fundamental defect in the application of these theories to surgical diagnosis.

In the final analysis, all real scientific progress must rest on the accumulated experience of many. Theoretical considerations, which are not based on and fully verified by experience, tend, by misleading, to retard a proper understanding of disease. Some of these postulates are undoubtedly true, but as always occurs in hypotheses not founded on experience, in these publications (one copying from another) certain factors present in surgical lesions of the brain were disregarded, viz., increased intracranial pressure, the acuteness and nature of the lesion, and the influence of the establishment of compensation.

All these factors fundamentally modify the objective reactions of the functional action of the brain—which the vestibular reactions primarily are.

Prior to 1914, having devoted considerable attention to the vestibular tests and having become somewhat discouraged by the diversity of the findings, I was not only impressed by these published accounts as truths by the facility with which they apparently clarified so many complicated problems, but also convinced that my failures were due to my inadequate knowledge. Not realizing their fundamental weakness, I abandoned my pre-conceived opinions, the result of experience, and adopted the published statements as demonstrated facts, only to find after many years that the statements were in many cases fallacious.

This has been the general effect upon the profession, as an examination of the literature of the past few years will demonstrate; for not only has little been written on the subject, but what has appeared deals in generalities of the most elementary nature. More significant still is the fact that while the reports of cerebro-surgical cases of brain tumors have enormously increased, few of them contain detailed records of the vestibular reactions; which forces the inference that the vestibular tests in cerebro-surgical conditions either have been generally abandoned, or have been of so little assistance as not to warrant their incorporation in the published report.

Physiological Considerations:

The vestibular reactions are, as we have seen, in many particulars very primitive reflexes of orientation, injury to which is followed by compensation for the reason that the more primitive the reflex the more diffuse and varied are the ways by which such impulses are transmitted; the reflex action being assumed by the nervous tissue as a whole. Thus, a man with a paralyzed arm from cortical destruction still gives a muscular twitch when a primitive protective reflex is stimulated, such as "auditory jump reflex," although the efferent tract necessary for voluntary movement is absent.

This probably explains why so many of the vestibular pathways have not been isolated, there being in fact no one definite bundle of fibres in many instances. In such a case all we are able to do is to study and attempt to interpret an absence or perversities of the whole or part of a normal reflex; and even in gross surgical lesions, in which large areas of cerebral tissue are affected, these perversities may not be present because of the establishment of compensation or because of the absence of definite cerebral localization of the function itself.

It is doubtful whether in very primitive reflex, any definite nerve tissue arc or pathway exists in the cerebral tissue, for we know that in spinal reflexes there is a "choice of efferent paths by which the excitation can be transmitted to the peripheral motor organ."*

Clinical Method of Interpretation Necessary:

If the vestibular tests are to occupy a place of practical value in the differential diagnosis of surgical and non-surgical lesions, I believe it is necessary that the present method of their interpretation from theoretical generalizations, based on supposed

*Luciana: Human Physiology, Vol. III., p. 93.

"interference with this or that" so-called "vestibular pathways", must be largely abandoned, and the abnormal vestibular reactions listed as they are clinically present in different diseases and lesions. It is only by some such method that definite information of clinical value can be obtained from the apparently confusing reactions.

In the present imperfect state of our knowledge regarding the vestibular reactions, the present method of attempted localizing intepretation based upon interference with vestibular pathways is as senseless as to argue that because "fever is a reaction to an irritant" whenever we have an irritant we must have fever. The presence or absence of fever is but one clinical manifestation of many diseases and lesions, and while of great diagnostic and prognostic value, it is of obscure origin.

If, however, the abnormal vestibular reactions are specially listed under a *clinical classification* (of lesions and diseases) as clinical symptoms (of such diseases and lesions); and data in a large number of cases obtained, will then, and only then, become possible to argue backward and to attempt cerebral localization by the assistance of the known vestibular manifestations. It is only by such a slow, laborious, and uninspiring method that real progress is possible.*

Clinical Classifications Under Which Abnormal Vestibular Reactions should be Enumerated and Discussed as Symptoms of:

(A) *Non-surgical Lesions, which because of their more or less* microscopic size are *not associated with increased intracranial pressure:*

(a) Chronic degenerative lesions; syringomyelia, disseminated sclerosis, locomotor ataxia, Friedreich's ataxia;

(b) Vascular disease; minute hemorrhages; small vascular occlusions;

(c) So-called functional nervous diseases, such as chorea, epilepsy, etc.

(B) *Surgical Lesions:* chief among which, because of their constant presence in all surgical lesions, should be enumerated;

(1) *The vestibular manifestitations of increased intracranial pressure in general:*

Having recognized that increased intracranial pressure, especially of the posterior fossa, gives rise to a distinct vestibular manifestation (which should be classed among the symptoms of increased intracranial pressure the same as papil-

*Eagleton, W. P.: Brain Abscess—Its Surgical Pathology and Operative Technic. Macmillan, 1922. (In press).

loedema), we can then further sub-divide the vestibular manifestations of surgical disease according to the origin and location.

(2) *Localized suppurative diseases of the posterior fossa;*

(a) Protective meningitis from labyrinth suppuration in intrapiarachnoid abscess;

(b) Intracerebellar abscess confined to the hemispheres of the cerebellum;

(c) Intracerebellar abscess invading the vermis, while the vestibular manifestations of:

(3) *Cerebral Abscess should be divided with those present into:*

(a) temporosphenoidal lobe abscess;

(b) frontal lobe abscess,

(c) occipital lobe abscess.

(4) *Surgical Non-suppurative Lesions* with increase of the intracranial pressure, do not lend themselves to analysis with the same facility as localized suppurative diseases, not only because they have not the same tendency that a brain abscess has for the selection of definite sites at the beginning, except in a few particulars, but also, *because* of the *chronic nature of their invasion,* they are not apt to give the clean-cut picture of vestibular manifestations induced by a localized area of acute suppuration; for while tumors (encapsulated) are frequently more circumscribed than brain abscess, compensation has had greater facility for re-establishment. Consequently, it is essential that they be listed separately from suppurative diseases.

The vestibular manifestations of brain tumors should be listed under those present as follows:

(A) *Tumors below the Tentorium:*

(a) Tumors of the acoustic nerve,

(b) Tumors of the pontine-cerebellar angle,

(c) Intercerebellar tumors of the cerebellar hemispheres,

(d) Tumors involving the vermis.

(B) *Tumors of the Cerebrum:*

(a) Pituitary growths,

(b) Suprasellar growths,

(c) Pineal tumors,

(d) Frontal lobe tumors, Anterior to the Rholandic fissue,

(e) Temporosphenoidal lobe tumors,

(f) Motor-tract tumors, Post Rholandic,

(g) Occipital lobe tumors.

I am perfectly aware of the many weaknesses of such a classification, but as it is based upon the usual lesions and their sites most frequently selected, it has some practical value, especially as during the period of invasion and before the establishment of compensation the vestibular manifestations are apt to be the most clear cut, and, consequently, of the greatest assistance.

Accidents During Performance of Vestibular Tests:

In addition to the possible danger of causing a recurrence of a previous purulent otitis by the application of a cold caloric, it should not be denied that the rotation test may, under certain conditions, precipitate alarming or even dangerous disturbances. This has occurred to me in two instances.

Case Reports

Case I: A girl of twelve years, prior to the evacuation of a temporosphenoidal lobe abscess, had a convulsion. The operation was followed by a small hernia which soon became completely covered with skin, and the patient was discharged as cured. Three years later she was recalled for examination, at which time there was a slight bulging about the size of half a marble over the site of the old hernia. The girl apparently had been in good health for three years. In the midst of the rotation she had a convulsion, became unconscious and was apparently dying. From this state she gradually recovered and was able to be returned to her home (about two hours later). When it is recalled that for three years she had had no cerebral symptoms, and that the only previous convulsion undoubtedly was due to her brain abscess, the inference is forced upon us that the rotation caused a severe disturbance, probably vascular, of her brain—made possible, perhaps, by the dural defect.

Case II: A woman, in her fifties, with a high blood pressure, developed a slight exophthalmos, which appeared first in one eye and then slightly in the other. At the time of the examination the veins of both ocular conjunctiva, especially of the left, were distended, as were the veins in both fundi, in which there were multiple small hemorrhages. R. V. 20/50; L. V. 20/70. There was a paralysis of the left external rectus. A distinct bruit could be heard all over her head, chiefly on the left side, which she said had been present off and on for the previous eighteen months, coming on suddenly and then gradually subsiding. (This and the diminished hearing in both ears, but especially in the right —the contralateral side—had been attributed to her high blood pressure.) The Weber was referred to the right. She gave a history of severe headache, accompanied by vomiting and swelling of her lids.

A diagnosis of pulsating exophthalmos from aneurismal varix of the internal carotid, with rupture into the cavernous sinus of the left side, was made. During her neurological examination it was discovered that she had a *spontaneous deviation* of the arm of *the contralateral side inward,* about two inches. I ordered that she be rotated. Rotation to the right; horizontal nystagmus, 24 seconds; no vertigo, but she vomited; rotation to the left; horizontal nystagmus, 23 seconds; no vertigo. Immediately following the rotation she had a severe pain in her head and began to vomit; both eyes protruded greatly, associated with such an intense oedema of the skin that the eyes could not be opened, and there were numerous fresh hemorrhages in both retina.

On the following day she was able to recognize hand movements in one eye, the other being totally blind except to light.

This attack resembled her previous attacks, but was much exaggerated. For several days I feared she would die. Remarkable to relate, during this time the pulsation in her head entirely disappeared, both subjectively and on auscultation, and never recurred.

Four days after the rotation I ligated the left common carotid artery in the neck. This was promptly followed by a recession of the eyeballs, and later by absorption of the hemorrhages.

One month later, R. V. 20/60; L. V. 6/200. The vision of the right eye —the contralateral—now began to fail; the conjunctival veins enlarged and fresh hemorrhages appeared. The hemorrhages were associated with an attack of glaucoma of the right eye, which rapidly caused her to become blind in that eye, while in the left—the primarily affected eye—the vision improved and today she has a vision in her left eye of 20/30.

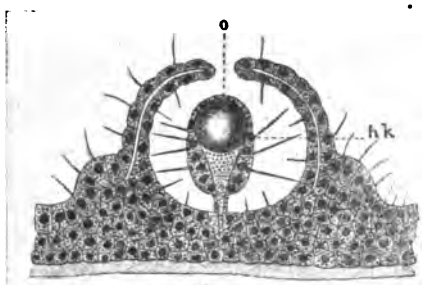


FIG. 239.—Auditory organ of the Rhopalonema still showing a small orifice (Hertwig).

hk, modified tentacle; *o*, auditory organ.

Figure 2. Vestibular Apparatus In Butterfly Showing Highly Developed Vestibular Apparatus But Absence of Auditory Apparatus.
From Morat, J. P.: Physiology of Nervous System. Keener, Chicago, 1906.

As in the previous case, it undoubtedly was an abnormal cerebral vascular system which caused the accident. There is, of course, no excuse for the latter case; she should not have been subjected to rotation nor a caloric, but with the former it shows that in vascular disturbances, either pathological or traumatic, rotation is not without some risk.

Biological Data of Clinical Significance about the Vestibular Apparatus:

Even in very low forms of life a well-developed vestibular apparatus is present, because orientation is necessary. In the invertebrate, life very early evolved a vestibular apparatus out of one of its most primitive attributes—viz., sensation; for "functionally, the vestibular apparatus is a transformation of the tactile apparatus adapted to analysis of pressure."* Fig. 2. It

*Morat, J. P.: Physiology of the Nervous System; 1906, p. 602.

was not until much later, as we have seen, that life, in the vertebrate, envolved an auditory apparatus for the further analysis of the waves of sound.

This fundamental evolutionary fact that the vestibular apparatus is of primitive origin, well developed in the invertebrate, and audition is of much later development, first appearing among the vertebrates, and then very rudimentarily, is of prime importance; for the simpler the form of life, the more easily is it able to dispense with its parts, or rapidly compensate for the loss of or an injury to them. This explains the ability possessed by man of rapidly compensating for, or entirely dispensing with, this rudimentary apparatus. It furnishes the clue to the clinical facts that the loss of or injury to the vestibular apparatus, both peripheral and central, is rapidly and even completely recovered from by the establishment of compensation, while damage to the auditory apparatus is permanent, there being no compensatory machinery to take its place. A man loses the hearing of both ears from suppuration—his vertigo, nystagmus and spontaneous pointing deviations soon disappear, but his hearing never returns.

During the past few years, as the wonders of recent discoveries regarding the vestibular apparatus have dazzled our intellect and stimulated our imagination, we have regarded the vestibular apparatus as highly specialized, on the same plane with speech, which possesses very definite "tracts", and an injury to which produces definite and irreparable manifestations. In this our error has originated. Because the vestibular apparatus is so *new to us*, we have treated it as new to Life, while in reality to Life it is one of the oldest attributes—not "as old as the hills," but much older; for when Life acquired it the waters covered the surface of the earth, and the hills were not yet.

A. Vestibular Tests in Non-surgical Lesions.

In a consideration of vestibular reactions the presentation of a clear-cut picture—a loss of such parts of otherwise normal vestibular reactions sufficient to warrant a vestibular peduncular symptom-complex or a vestibular posterior longitudinal symptom-complex—must presuppose a very small lesion, so small, in fact, as to be almost microscopic. In multiple sclerosis, syringomyelia and Friedreich's ataxia the vestibular reactions are of the greatest localizing value because the lesions are degenerative and microscopic in extent, without a compli-

cating increase of intracranial pressure. Only in such lesions will the rules formulated for "interference with such and such a vestibular pathway" find a proper application.

B. VESTIBULAR TESTS IN SURGICAL CRANIAL LESIONS.

Visualization of the Lesion—not "Interference with a Vestibular Pathway"—Necessary for Surgical Localization:

In considering the vestibular reactions in their relation to surgical lesions of the brain, the surgeon should not regard the intracranial portion of the vestibular as definite inflexible tracts, which are either opened or closed as an electric circuit. Unfortunately, surgical cerebral pathology is not so simple.

The surgeon must think of something which can be definitely visualized—an isolated area, such as an endothelioma, cyst, or extradural hemorrhage, or a portion of the brain surrounded by a zone of induration, such as an abscess or an infiltrating glioma.

All surgical lesions cause an increase in the intracranial contents, and give rise to profound disturbances in all parts of the intradural tissues, influencing the vestibular reactions along with the rest, although the growth or abscess may not be situated directly in the course of any vestibular neuron. Consequently, when we attempt to apply the rules of vestibular tract localization, (which from a pathological standpoint may be accurate), the surgeons must not leave out of consideration the vestibular manifestations of the increased intracranial pressure itself, which not only modify but frequently overshadows the other vestibular manifestations.

Factors Influencing and Altering the Vestibular Reactions in Surgical Lesions:

I. Increased intracranial pressure,

II. Establishment of compensation in the two vestibular apparati,

Corollary Factors:

III. Duration of the lesion—whether acute or chronic,

IV. Nature of the lesion—whether causing symptoms (a) by pressure alone, as in endothelioma, cysts, intradural or intrapial hemorrhages, or (b) by destruction or infiltration, abscess or infiltrating tumors.

(a) The Vestibular Manipulations of Increased Intracranial Pressure.

Through ignorance of the work of other observers, the vestibular manifestations of increased intracranial pressure have been largely disregarded. *The vestibular manifestations of increased pressure, especially of the posterior fossa, are definite,* and so outspoken as to overshadow, and in the majority of cases, to prevent the localization of surgical lesions by the vestibular tests alone, even when an intracranial portion of the vestibular apparatus itself is directly involved, as in many cases of large cerebellar abscess; for *in surgical lesions* it must be realized that the *vestibular manifestations which stand out* most prominently are *the manifestations of increased intracranial pressure,* and these vestibular manifestations are disclosed only by the caloric tests, just as the ocular manifestation of increased intracranial pressure—papilloedema—is disclosed only by the ophthalmoscope.

A large personal experience would lead me to postulate that in the presence of increased intracranial pressure the vestibular reactions are of *great general diagnostic value, although they are but rarely useful in cerebral localization.* In my opinion, they should take, like the demonstration of a papilloedema by the ophthalmoscope, their position as of great clinical assistance in the diagnosis of increased intracranial pressure, especially of the posterior fossa.*

In the year 1912, in a communication to the American Otological Society†, after recording the history of three cases of tumor of the brain involving the posterior fossa, and commenting upon *the absence of reaction to the cold caloric when applied to the ear of the opposite side of the lesion* in all three cases, I stated; " This condition, if generally present, may be of service in *diagnosing increased intracranial pressure* involving the auditory apparatus, possibly at an early stage," and I concluded with the statement: "A study of these three cases leads me to believe that in *intracranial lesions of the posterior fossa there may*

NOTE:—Prior to the publication of this paper I had applied the cold caloric in the upright position, thus stimulating the vertical alone. I had no knowledge that in cases of increased intracranial pressure of the posterior fossa, a vestibular reaction follows when the opposite horizontal canal is stimulated.

*It is strange that this error has been made, because of the former fallacy regarding the localizing importance of papilloedema.

†Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin; Report of Three Cases with a Previously Undescribed Aural Condition. Transactions of the American Otological Society, June 10-11. 1912, Vol. XIII, Part III, p. 564 Also, Laryngoscope, 1913, Vol. XXII, p. 592.

occur early reduction of the excitability of the vestibular apparatus, not only of the affected side but of the unaffected side as well.

Prior to the appearance of my paper there was no recorded observation, although Neumann previously had called attention to the reduced irritability to the cold caloric of the contralateral side in suppurative otitis. Consequently, it is claimed that my observation was the first recorded vestibular manifestations of increased intracranial pressure, and more important still, that at that time *the first proper interpretation* was offered.

Since 1912 the accuracy of this vestibular manifestation of increased intracranial pressure has been substantiated in so many instances, both clinically and at post-mortems, that I have come to regard it as of the first importance. In my opinion, it is the failure to appreciate its uniformly overshadowing presence that accounts for many of the disappointments and for much of the scepticism in regard to the value of the reactions in surgical lesions.

Because of its great importance to the clinician credit for whatever value the description and interpretation of the reactions possess is due to this early observation.

The vestibular manifestation of increased intracranial pressure, viz., the absence of reactibility to the cold caloric on the opposite side, is frequently mentioned in the cases reported by other observers, but its diagnostic value apparently has not been appreciated. During the past few years this vestibular manifestation of increased intracranial pressure has been elicited by me in a large number of cases of various intracranial pathological processes. In numerous instances its reliability has been proved by a lumbar puncture; and on several occasions an amber colored fluid under pressure—showing a pathological condition—has been encountered in cases in which the routine otological examination had led me to believe a peripheral lesion was present.

I can confidently affirm that the demonstraation of an increased intracrainial pressure by the caloric has largely revolutionized my conception of many of the poorly understood intracranial lesions, such as cases of deafness, psychoses, and headaches following trauma, and in several instances as well has rendered possible a diagnosis of probable brain tumor long before the appearance of a papilloedema.

Case III. THE VESTIBULAR MANIFESTATIONS OF INCREASED INTRACRANIAL PRESSURE, DIAGNOSING A PROGRESSIVE DEAFNESS AS OF INTRACRANIAL ALTHOUGH APPARENTLY OF PERIPHERAL ORIGIN; LUMBAR PUNCTURE VERIFYING THE DIAGNOSIS.

P. G. Male, 29 years of age.

Outstanding Symptoms:

Increasing deafness in both ears, especially the right, during the past five years; tinnitus in both ears; severe headaches.

Family History in regard to hearing—negative.

History:

No preceding illness. For six years he has had loud noises in his ears, and has been growing deaf gradually in both ears, with headaches and slight dizziness in moving or turning his head. He never had pain in nor discharge from his ears. The conditions seem to grow worse each winter but remain stationary in the summer. He hears better in quiet surroundings and on clear days. The tinnitus in the right ear resembles escaping steam; in the left, a booming sound. The patient believes deafness was noticed before the tinnitus. One year ago his tonsils were removed; within the last year he has had two operations on his nose—all to improve his hearing but without benefit.

Examination: O. E. negative. *Vision* normal with proper correction. *Mouth*: Teeth showed the gums of upper jaw red high up near the fold of the membrane. X-ray of teeth negative. *Nose*: The left inferior turbinate seemed to run far back and hang low posteriorly; membrane glossy; no pus. Transillumination showed no definite shadow but was not bright on either side. Blood pressure 128 over 90.

Hearing: Watch heard at one foot; whisper at five feet; voice across room.

	R.		*Weber.*
A. C.	40	seconds	Head; both ears same; Teeth Left. Chin, Left.
B. C.	110	seconds	
			Rinne.
	L.		
A. C.	65	seconds	Head: Hears same in both ears, makes no difference which ear is plugged. Teeth: Same as head. Chin: Best in Left ear.
B. C.	115	seconds	

Vestibular Reactions:

Chart I. P.G.

R

Spontaneous

Nystagmus - 0
Past-pointing - 0
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus - → 46 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

Rotating to Left

Nystagmus - ← 44 seconds; good amplitude.
Past-pointing - +
Dizziness - +
Nausea - +

October 11:

R

Cold Caloric in Right

Vertical

Nystagmus - ↷
Past-pointing - 0
Dizziness - ++
Nausea - +

Horizontal

Nystagmus - →
Past-pointing - 0
Dizziness - +++
Nausea - (vomiting) ++

Cold Caloric in Left

Vertical

Nystagmus - 0
Past-pointing - 0
Dizziness - +
Nausea - 0

Horizontal

Nystagmus - ← +++
Past-pointing - 0
Dizziness - ++++
Nausea - ++

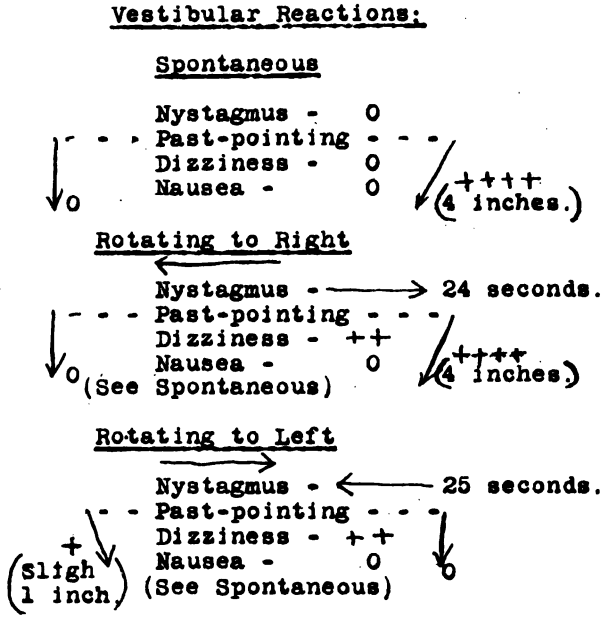
Blood Wassermann Negative. *Lumbar Puncture* gave fluid under *very great pressure, opaque, of a slightly amber color;* globulin and sugar normal; cell count 6.5 per cmm.

Chief Complaint: Progressive loss of hearing, tinnitus with headaches.

Abnormal Findings: Exaggerated patella reflexes; great exaggeration of the duration of the nystagmus; lack of proper pointing deviation; positive Rhomberg. "Left caloric reaction gave no nystagmus, some dizziness, no past-pointing; consequently, the trouble probably is a right-sided lesion."

Diagnosis: "In view of all the symptoms it would seem possible that the man had a disseminated sclerosis. It sounds like a toxic lesion; possibly a thyroid case. The amber-colored cerebrospinal fluid is diagnostic of a degenerative lesion."

January 28:



Increased Intracranial Pressure a Handicap in Localization by Means of the Vestibular Reactions:

Surgeons and clinicians should appreciate that while in a microscopic lesion involving the vestibular tract, exact localization by the vestibular reactions of the site of the lesion, either in the cerebellum, the cerebellar peduncles, the posterior longitudinal bundle, or even in certain parts of the cerebrum, is frequently possible; the presence of increased intracranial pressure of the posterior fossa, causing as it does a uniform absence or reduction of irritability of the vertical canals of the

opposite side, as well as those of the affected side, generally prevents accurate localization by the vestibular tests alone. Much in the same way as an intense papilloedema, by causing a general contraction of the visual field, may obscure a localizing hemianoptic defect (in the visual fields).

In cases of increased intracranial pressure, the reduction of the irritability of the vertical canals of the opposite as well as those of the affected side renders diagnosis impossible by the vestibular manifestation alone.

(b) Establishment of Compensation in the Vestibular Mechanism-Cerebral Reduction of the Duration of the Induced Nystagmus on Turning:

Another factor of fundamental importance, which further interferes with the localizing value of the vestibular reactions and greatly complicates their interpretation in lesions affecting the intracranial portions of the vestibular apparatus, both gross and microscopic, is the early establishment of compensation. To this I also called attention in the paper referred to, as shown in the reduction of the duration of an induced nystagmus and of the caloric excitability of the tracts of the two sides. This readjustment, to which I gave the name of "establishment of compensation", is intimately associated with and allied to the vestibular manifestations of increased intracranial pressure.

It may be imperfectly illustrated as follows: With a normal vestibular apparatus—end-organ and central—stimulation of a horizontal canal by turning produces an induced horizontal nystagmus of 24 seconds' duration, 16 seconds being contributed, it is believed, by the horizontal canal of the labyrinth chiefly stimulated and 8 seconds by the opposite labyrinth.

If one labyrinth is destroyed the maximum duration of induced nystagmus obtainable is 16 seconds, wholly contributed, of course, by the remaining intact labyrinth, while 8 seconds of nystagmus occur on turning in the opposite direction, this 8 seconds also coming from the remaining labyrinth.

In time, however, the duration of the nystagmus is again equalized—although one labyrinth continues out of commission—by, I believe, the establishment of compensation. The duration of the induced nystagmus on maximum stimulation by turning toward the destroyed labyrinth, which immediately after the labyrinthine destruction was 16 seconds, is reduced to 10 or 11 seconds; and the stimulation—on turning away from the lesion—which immediately after the labyrinthine destruction had a duration of 8 seconds, now is increased almost to the same as that recorded in turning in the opposite direction, viz., to 10 or 11 seconds. In other words, a compensation has been established by dividing the duration of the induced nystagmus equally between the two sides, although one end-organ is now contributing all the stimulation.

C. VESTIBULAR TESTS IN DIFFUSE SUPPURATIVE AND HEMORRHAGIC DISEASES OF THE MESHES OF THE PIARACHNOID OF THE POSTERIOR FOSSA.

Even with these handicaps the vestibular tests, if routinely and conscientiously applied as part of the general neurological examinaton—just as the hearing and sight are tried and the eye grounds inspected—will be found to be of the greatest assistance and will, as our knowledge of the vestibular apparatus is amplified, become of more and more assistance in localization. Progress, however, can be made only by the publication of case reports, so that large numbers of detailed reports may be collected and properly analyzed.*

D. VESTIBULAR TESTS IN PROTECTIVE MENINGITIS OF THE POSTERIOR FOSSA FROM LABYRINTH DISEASE OR INTRAPIARACHNOID ABSCESS.

With this in view the following four cases are presented in detail. From them certain deductions are to be drawn. Whether, however, these deductions will be substantiated must depend upon the observations of many independent investigators.

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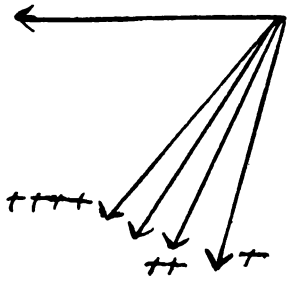
*In the publication of reports of vestibular findings, all details must be incorporated, as the lack of minute details—such as omitting to state the exact position of the patient, whether lying in bed or siting with the head forward at 30° during the application of the cold caloric—may invalidate its value.

For the past-pointing deviations the following graphic scheme may be used: Each + represents one inch until four ++++, after which the angle should be used.



For accurate recording of details I would advocate following routinely the scheme of examining and recording as suggested in the Guide to a Detailed Neurological Examination in "Fracture of the Skull with Chart of Clinical Classification and Guide to Detailed Neurological Examination", Eagleton, W. P., Archives of Surgery, July, 1921, Vol. III, pp. 140-153, in which an attempt is made to compel attention to details both during the examination and when recording them; while for purposes of rapid and visualized comparison, such as induced or spontaneous past-pointing and nystagmus, the author offers the visualizing charts given with the case reports in the present paper.

REPORT OF FOUR CASES OF PROTECTIVE MENINGITIS FOLLOWING LABYRINTH SUPPURATION OR TRAUMA, PROBABLY ASSOCIATED WITH A LOCALIZED COLLECTION OF CEREBROSPINAL FLUID OVER THAT PORTION OF THE ANTERIOR SURFACE OF THE CEREBELLUM WHICH LIES BEHIND THE POSTERIOR SURFACE OF THE PETROUS PYRAMID; WITH A SUGGESTED DIAGNOSTIC VESTIBULAR SYMPTOM COMPLEX—NAMELY, (1) ABSENCE OF COLD CALORIC OF THE VERTICAL CANAL OF THE UNAFFECTED SIDE (FROM INCREASED INTRACRANIAL PRESSURE), AND (2) IMMEDIATE REDUCTION BY 50% OF THE MAXIMUM DURATION OF THE INDUCED NYSTAGMUS ON TURNING, (WHICH WOULD BE OBTAINABLE WERE THE LABYRINTH ALONE AFFECTED; WITH (3) EVIDENCE OF THE INVOLVEMENT OF A PAST-POINTING CENTER IN THE CEREBELLAR CORTEX—AS THE POINTING DEVIATIONS COULD NOT BE ALTERED IN DIRECTION UNDER ANY FORM OF STIMULATION.

Case IV. CASE OF SEROUS MENINGITIS WITH PAPILLOEDEMA, CLOUDY FLUID, HIGH CELL COUNT, FOLLOWING LABYRINTHITIS; AND SINUS THROMBOSIS WITH ABSCESS OF THE JUGULAR VEIN; ASSOCIATED WITH SPONTANEOUS PAST-POINTING DEVIATION OF BOTH ARMS AWAY FROM THE LESION, WHICH COULD NOT BE ALTERED BY STIMULATION, ABSENCE OF OPPOSITE VERTICAL CANAL TO COLD STIMULATION (FROM INCREASED INTRACRANIAL PRESSURE)—AND IMMEDIATE REDUCTION OF DURATION OF NYSTAGMUS TO EIGHT SECONDS.

(Successive charts show the persistence of the pointing deviation until altered by the opposite ear becoming affected, when there was a slight alteration.)

August 19, 1919, J. B., man, twenty years of age.

History: Has always had running left ear with frequent attacks of pain. Always has been subject to headaches. Four weeks previously had pain in the ear—a usual condition. The following day had a severe chill, followed by fever, then a sweat. This was repeated several times during the next four weeks, with considerable headache. Admitted to Newark City Hospital. Examination disclosed chronic mastoid involvement. On opening the mastoid Dr. E. A. Curtis found a large septic clot in the sinus. Condition became very poor during the operation; man thought to be dying. Returned to bed without having ligated the jugular. Did well for a few days, then recurrence of chills and pain. About ten days later I exposed his jugular; found an abscess filling the whole of the interior of the vein itself. *Free pus in the vein.* It was not in the fascia surrounding it but in the vein. On slitting the vein, free pus liberated——no clot. Followed downward and in the lower portion found a clot. Incision in vein went below the level of the clavicle, but did not cause bleeding. Jugular bulb now exposed. Free bleeding obtained from the upper portion of the lateral sinus.

Examination of his eyes prior to the second operation showed no change in the discs. Immediately after the second opration the man complained of dizziness, which continued. Did fairly well, although he lost considerable flesh. Did not look sick enough to warrant any further interference.

Seven weeks after the original involvement, and ten days after the second operation, man had two chills, followed by a period of violent headaches, located in frontal region; headaches would pass away as suddenly as they came. Temperature not much elevated. Examination now revealed double papilloedema. (How long this had been present was not known. It was not present prior to the second operation.) His labyrinth now entirely dead. Had no spontaneous nystagmus, but a *spontaneous pointing deviation of both hands to the right,* (away from the lesion). Cold caloric on left (affected) side produced no nystagmus, no vertigo—dead labyrinth, lost flesh. On walking, would fall to the right side (away from the lesion). Patella reflexes present. Had a slight area of anesthesia—only partial—around the third branch of the tri-facial. Had one severe attack of vomiting. Lumbar puncture revealed fluid cloudy, under pressure. Cell count 160; increased globulin. Repeated blood cultures—all negative. No Romberg.

Deductions from Examination:—"It would seem from the pointing deviation to the right side, falling to the right side—both way from lesion—that the inward pointing center (Bárány's) of the cerebellum opposite to the posterior surface of the petrous portion of the temporal is involved. This is also confirmed by the cerebrospinal fluid, giving a protective meningitis with a slight area of anesthesia. If we rotate him and cannot produce a pointing deviation of the left, this may be confirmed."

Sept. 29, 1919. 1—Rotation to right: No nystagmus; no dizziness; no nausea; no deviation of right arm; slight deviation of left to right. 2—Rotation to left: Nystagmus to right, of eight seconds; very slight dizziness; no nausea; deviation of right arm two inches to left; no deviation of left.

Third Operation: Incision of dura over anterior portion of cerebellum; evacuated considerable amount of turbid cerebrospinal fluid; no pus; exploration of cerebellum negative.

Uneventful recovery followed operation.

Subsequent History: May 13, 1920. Right ear has been discharging for some time; during the past two weeks left ear has also been discharging, and man had aching pain back of the ear. Right ear contains large granulations, occluding the canal. Very deaf. Sudden left facial paralysis a few days ago. At times some blurring of vision. O. E. negative BS 20/20.

Right.	Left.
Watch, three feet	Watch, not heard
Whisper, four feet	Whisper, not heard
Voice, across room	Voice, not heard

Rotating to Left—Has nystagmus to right of 9-10 seconds; no dizziness. No deviation of right; left, deviated to *right.* (Tried twice.) (See spontaneous pointing.)

Rotating to Right—Head forward; did not fall; no dizziness.

Rotating to Left—Head forward, did not fall; no dizziness.

Cold Caloric in Right—*Head Erect;* produced no nystagmus or pass-pointing; no dizziness; no nausea; (increased pressure).

Cold Caloric in Left—Did not produce nystagmus; past-pointing; (Dead Labyrinth) dizziness or nausea; with head erect or back.

May 13, 1920:

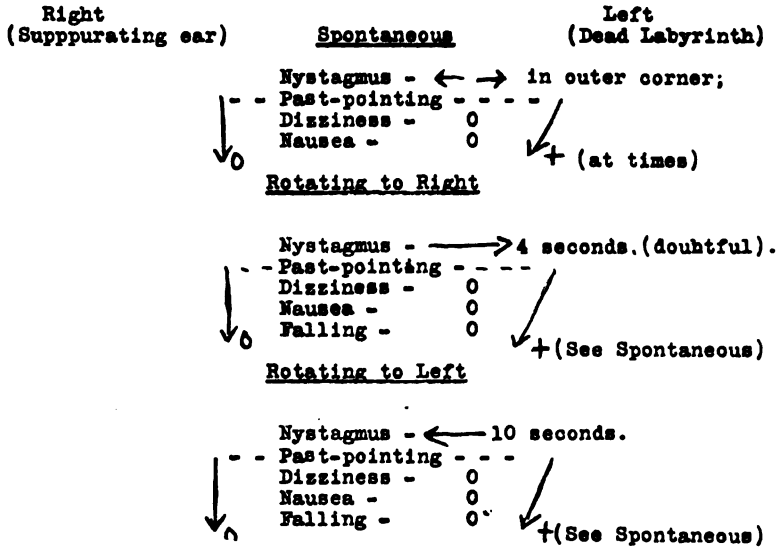
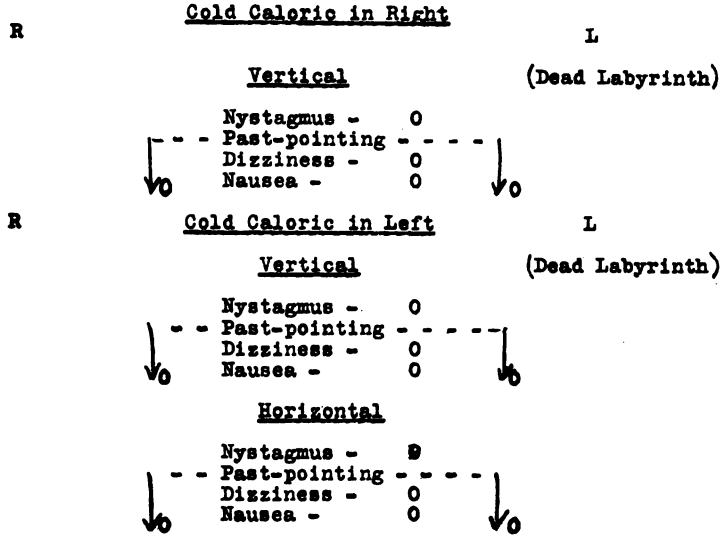


Chart I. J.B.

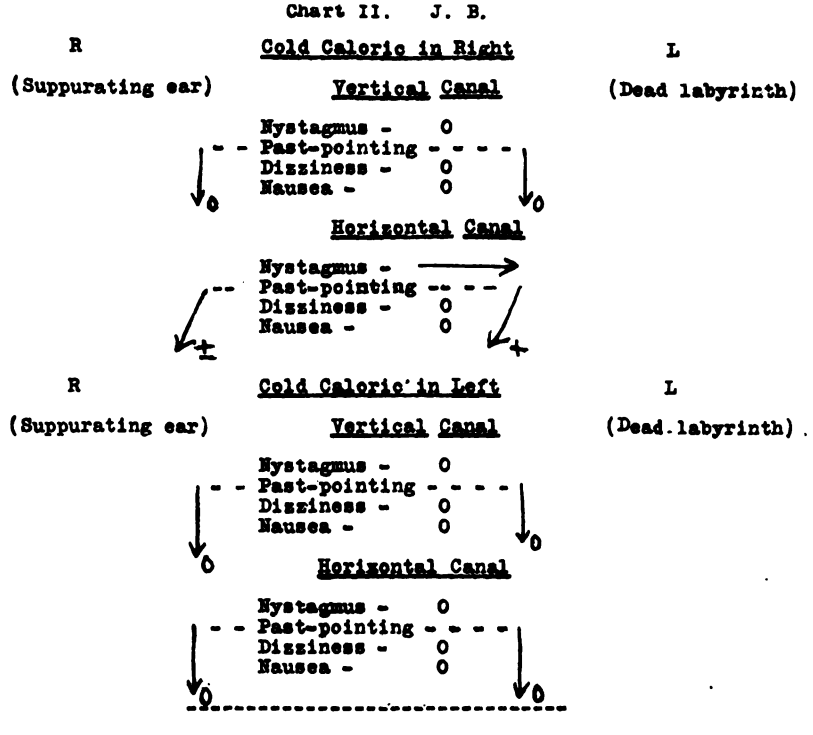


Shortly after the above observations a simple mastoid operation was performed on the right ear.

June 11, 1921. The right ear had continued to discharge from the wound until about three weeks ago, when a large piece of bone came from back of the ear; since then the discharge has ceased. Patient felt well until four days ago; then, sitting down quietly at home, seemingly in usual good condition, he suddenly, on standing up, had severe dizziness, a tendency to fall to the right—which has continued—and nausea at first. The dizziness is not quite so severe now; it came on with a sudden closing of his right ear. The left ear is very deaf following the mastoid on that ear; he has a noise like a faint whistle in the left ear; no pain in ears; dopey feeling in head. Blood pressure 102-68.

Outstanding Symptoms: Sudden closing of the right ear and at once severe dizziness; hearing reduced in that ear. Blurring vision in walking since present dizziness, which clears when he is not in motion.

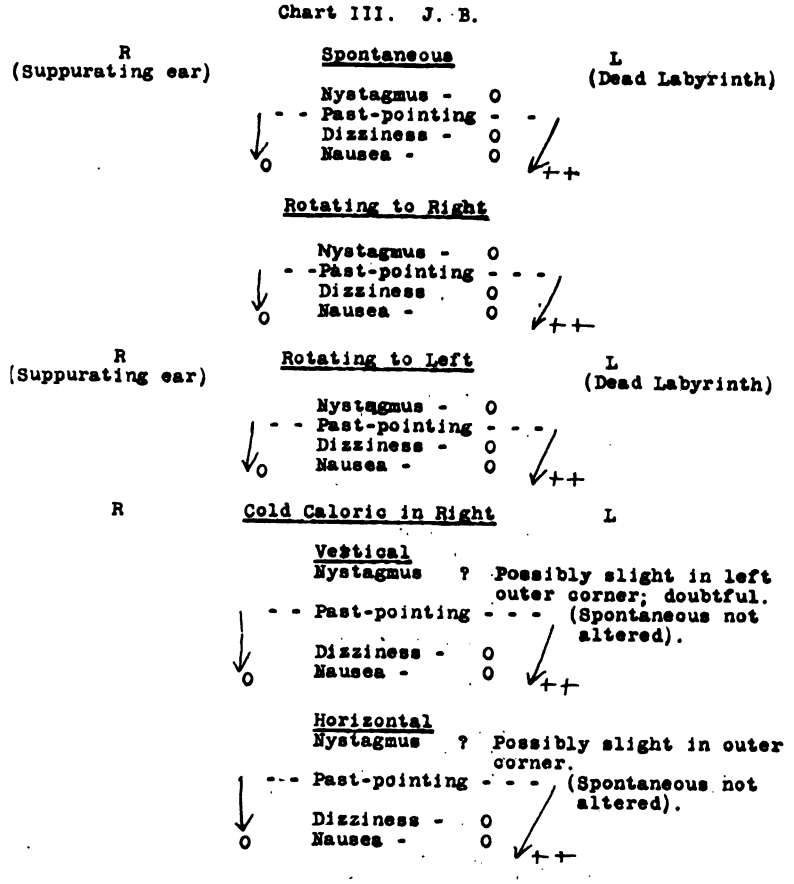
Chart II. J. B.



August 19, 1921. Has become totally deaf; is very dizzy and complains of pain over left eye. Sight is reducing. A discharge, with odor, from right ear for two weeks. Falls or staggers at once on attempting to rise, from dizziness and in falling *goes to left side.*

Outstanding Symptoms. Lately totally deaf; discharge with odor from right ear; severe dizziness; general weakness; pain over left eye.

Right Ear. Hears with noise box in left ear, but words must be shouted. Slight discharge. Granulations at site of drum.



Case V. Case of Acute Suppurative Labyrinthitis Followed by Serous Meningitis Associated with Vertical Nystagmus; Paralysis of Superior Oblique of Homolateral Side; Slight Facial Paralysis and Deafness of Homolateral Side; Homolateral Arm Slightly Incoordinate; Spon-

TANEOUS POINTING DEVIATION OF BOTH ARMS TO SIDE OF LESION; FLUID CLEAR—UNDER SLIGHT PRESSURE—BUT ELELVATED CELL COUNT; REDUCTION OF DURATION OF NYSTAGMUS ON TURNING.

B. R., History: Earache in left ear following an attack of tonsillitis early in April. Two days after earache the ear opened voluntarily and discharged more or less continually for two weeks.

April 16. An aural consultant diagnosed slight mastoid involvement. Paracentesis performed. April 28: the ear stopped discharging; vomiting and other "gastro-intestinal" symptoms followed. Double vision appeared, one object being seen about four inches lower than the other. Another consultant diagnosed "gastro-intestinal trouble." After bowel movement patient improved. This improvement lasted until following evening, May 1st, when another attack of pain in stomach occurred, and headache; followed by period of improvement until May 4, when there was recurrence of headache and "sick stomach" which again gradually subsided.

May 9. Became lanquid, vomited, and complained of severe headache. Diagnosis of acidosis.

May 10. Patient remained languid; became sleepy, and vomited twice. Another consultant—Dr. Eugene Murray called. Diagnosis, meningitis.

May 11. Patient admitted to hospital. Complained of constant temporal and occipital headache, with pain in neck; deafness of left ear for past three weeks, with tinnitis and dizziness. (There had been no chill and temperature was but slightly elevated.)

Examination. "Nystagmus, vertical, especially upon looking to the left. Possible paralysis of external rectus of left eye; left eye lower than right, left pupil larger than right. Total deafness of left ear; left drum normal, no tenderness over mastoid. Slight left-sided facial paralysis." Has lost flesh. Tongue protrudes in median line. No difficulty in swallowing.

Vestibular Manifestations. Vertical nystagmus; spontaneous pointing deviation of both arms to left; left arm co-ordinates imperfectly.

Lumbar Puncture. Spinal fluid clear, under very slight pressure; sugar reduction normal; globulin slightly increased; cell count 60 per cubic mm. No organisms.

Pre-operative Diagnosis. Labyrinthitis, with cerebellar involvement. Abscess?

Operation. Drainage of diseased deep cells of petrous pyramid; opening of semicircular canals and cisterna of internal auditory meatus. Incision of dura over cerebellum.

Operation Notes. The mastoid was opened and found full of granulation tissues; no pus; the solid angle between sinus and petrous bone opened; small, deep cells found involved and filled with granulation tissue but no pus; proceeded inward behind posterior and semicircular canals. No bleeding. Was compelled, because of forward-lying sinus, to chisel away posterior arm of posterior semicircular canal, which exposed both canals well; canals contained no pus and no fluid. Pin-point opening found in dura (which may have been operative). Discharge of cerebrospinal fluid on reaching internal auditory meatus. Dura over this area covered with granulations.

At completion dura was very black; two or three openings in dura from which cerebrospinal fluid was flowing, but not in great quantity. Middle fossa not exposed; sinus not injured; middle ear was not opened.

Operative Comment. "It would seem that this child has a serous labyrinthitis—possibly a brain abscess *over* the cerebellar region. It might have been better if the labyrinth had been opened through the middle ear and the cochlea exposed as well as the semicircular canals. Exploration into cerebellum was not attempted, although the spontaneous deviation—the lack of good coordination of the same side—pointed to an involvement of the brain stem."

At completion of operation the child apparently could not move its arm on that side, but this rapidly disappeared. O. E. was negative.

May 12. Temperature 100-99°; pulse very fast, 118-148, and at times irregular.

May 13. (Second day after operation). Pulse still rapid, 130; temperature 99-101°. Patient lies on right side, in which position eyes are deviated toward the right and downward; ophthalmoscopic examination negative, but may be slightly indistinct. Complains of soreness of throat. Slight facial paralysis, about the same as before operation. Left arm moves well, but incoordinately, Leucocyte count 19,200. Polynuclears, 85%; large Lymphocytes, 9%; small Lymphocytes, 5%.

May 16. Nurse reports "She seems to look at you with the right eye." Left pupil larger than the right. O. E. negative, but both discs whitish. Paralysis of left external rectus. Patient has tendency to look downward and to the right, in which position of eyes there is no nystagmus; there likewise is no nystagmus when looking downward and to the left, but rotary nystagmus on looking upward when patient is in recumbent position. Tongue protrudes in median line; palate goes up on right side; patient complains that when she swallows the solids and liquids "do not go down right". There seems to be a shoulder girdle in which the inco-ordination is most marked when she has shoulder and elbow free. Both vocal cords move normally.

May 18. O. E. negative; temperature normal; pulse still fast—118-130.

May 19. Smear and culture from left ear; streptococci and cocci probably of sarcina group.

May 29. Left pupil larger than right; left eye turns in on looking to right; rotary nystagmus to right, but absent on looking to left when the eye is still (this occurs when either eye is covered). Complete paralysis of the left external rectus. Incomplete facial paralysis. Marked rotary nystagmus in looking upward; left eye slightly lower than right. No dizziness when patient tries to sit up, but in returning to recumbent position patient becomes dizzy.

June 7. O. E. negative. The left disc is whiter than the right, but it may be that the right is too red. Incoordination of the left arm. Left complete facial paralysis; cannot close eye; complete paralysis of external rectus; on closing left eye moves right eye inward without nystagmus, on turning it outward to the left there is nystagmus. With both eyes open, on looking upward there is rotary nystagmus of vertical tendency; no nystagmus on moving to left, but of course the left eye does not go outward because of paralysis of left external rectus; left eye is downward, and on moving, raises the upper lid; looking to right, no nystagmus. No nystagmus on looking downward, but marked vertical nystagmus on looking upward. Apparently has a paralysis of the superior rectus or of the superior oblique—I think of the superior rectus, but I am not sure. Temperature, normal; Leucocyte count, 9,000; Polynuclears, 67%; large lymphocytes, 7%; small lymphocytes, 25%; eosinophiles 1%.

June 8. *Vestibular Reactions Spontaneous.* "No spontaneous past-pointing; spontaneous rotary nystagmus to right at short intervals; the nystagmus is more noticeable when the patient looks to the right and somewhat upward. It is more constant in this position than when she looks straight ahead; it is also more marked when she looks upward. When she looks straight ahead the act of blinking seems to increase the nystagmus."

Rotating to Right. (1) Right hand deviated two inches to right—did not deviate more than once. Left hand did not deviate. Dizziness; no nausea. (2) No deviation of either hand. No perceptible nystagmus was induced.

Rotating to Left. No deviation of either hand; horizontal nystagmus to right of 8-10 seconds. Dizziness. No nausea.

Cold Caloric in Left. Head erect; produced no reaction. (Dead labyrinth). Head back; no perceptible nystagmus induced, tho possibly slightly horizontal to left. Dizziness. No past-pointing.

August 14. Double vision improving. Images 6 inches; at times vision is single. Image separates on looking to the left. The child apparently is perfectly well. Wound healed.

Case VI. Case of Generalized Protective Meningitis, Result of Suppurative Labyrinthitis, with Vestibular Manifestations of Increased Intracranial Pressure—Non-Reactibility of Opposite Vertical Canal to Cold Caloric—From Which a Diagnosis of Increased Intracranial Pressure Was Made; Reduction of Duration of Nystagmus on Turning; Inability to Cause Arms to Deviate Toward Side of Lesion; Lumbar Puncture Revealed Pressure and High Cell Count—Following Operation Severe Symptoms of Meningitis.

J. G.: male; adult. Seen with Dr. Baldwin November 24, 1919.

History. Had discharging right ear for three months. For the last three weeks had pain in right ear with severe pain in head, in right temporal region; attacks of dizziness during past three weeks—"will be going along and suddenly something occurs and I sway. I have never fallen but have run into things." Does not know to which side he sways. Several chilly sensations and two attacks of vomiting. Last two days facial paralysis.

Examination. Right drum membrane destroyed; granulations and pus in external canal. No hearing in right ear.

Rotating to Right. Head forward; on straightening up, fell to right; dizzi- No dizziness or nausea. Wide deviation of right to right (10 inches). Slight deviation of left to right (3 inches).

Rotating to Left. Horizontal nystagmus to right of *five* seconds. No dizziness or nausea. No deviation of either hand; quick movement and large amplitude; wide pointing deviation of both hands to left; dizziness.

Falling.

Rotating to Right. Head forward; on straightening up, fell to right; dizziness and nausea.

Rotating to Left. Head forward. Did not fall; very slight dizziness.

Cold Caloric in Right. Dead labyrinth.

Cold Caloric in Left. Produced no nystagmus—if any it was horizontal to left—no pointing deviation of either hand, no dizziness or nausea.

Throwing Head Back. Produced a violent horizontal nystagmus to right. Temperature 99°. Pulse 76.

November 27. First lumbar puncture performed; fluid clear, under pressure. Globulin increased. Fehling's slightly reduced. Cell count 100 cm. No organisms.

November 28. Second lumbar puncture; fluid clear, under pressure; globulin increased; sugar reduction not normal; no organisms.

November 29. Operation. Labyrinth opened; fistula found; granulations. Neumann's operation. Posterior fossa opened behind posterior pyramid; a quantity of cloudy cerebrospinal fluid evacuated.

Following operation patient had severe pain over left eye, numbness of fingers of left hand; was restless at night; temperature 101°-103°; Leucocytes count, 12,800; Polynuclears, 73%.

December 3. Stiff neck; severe pain in head; Leucocyte count, 16,400; Polynuclears, 85%; smear from wound, staphylococcus, streptococcus mucosus capsulatus. Temperature 102°-104°; pulse slow 82-90.

From December 3 to December 28 patient continued with restlessness at night, slightly delirious with alternating attacks of stupor, from which he could easily be aroused; severe pain in head; stiffness of neck; Kernig; pain behind left eye; photophobia, with a continuously high temperature—101°-105°—and a proportionally slow pulse. Several blood cultures were negative. Leucocytosis.

December 26. Third lumbar puncture; no pressure; color pale yellow; globulin greatly increased; sugar reduction, about one-half normal; cell count, 633 cm.

January 7, 1920. Patient looked very ill; pulse very fast and of poor quality; but rested better at night; still complained of stiffness in back and legs, but head felt better.

January 18. Patient much better in every way; temperature normal.

February 24. Patient discharged from hospital feeling fairly well but with a discharging ear. A few days later he discharged a sequestra of cochlea, after which the ear promptly healed and all symptoms disappeared.

A similar case with dead labyrinth, high temperature, delirium, almost maniacal headache, double papilloedema, cloudy spinal fluid, with cell count of over 3000 but without microörganisms, an exenteration of the labyrinth, with incision of the dura, was promptly followed by recovery.

Case VII. RESULT OF CLEANING OUT DEEP CELLS OF PETROUS DEVELOPED LABYRINTHITIS AND SEROUS MENINGITIS WITH HIGH CELL COUNT OF LUMBAR PUNCTURE FLUID; SPONTANEOUS ROTARY NYSTAGMUS AND INWARD POINTING DEVIATIONS OF BOTH ARMS, THE DIRECTION OF WHICH COULD NOT BE ALTERED. (1) ABSENCE OF REACTION TO COLD CALORIC OF OPPOSITE VERTICAL CANAL (FROM INCREASED INTRACRANIAL PRESSURE) AND (2) REDUCTION OF DURATION OF INDUCED NYSTAGMUS TO FIFTEEN SECONDS GIVING RISE TO THE SUPPOSITION THAT THE FUNCTIONAL TEST ULTIMATELY WOULD RETURN TO NORMAL.

Boy, G. C., seventeen years of age. History. February 22. Scarlet fever, and following this, about February 28 left side, earache with discharge, during which time patient had delirium, and at least one chill. During discharge, had headache over temple. Followed by period of improvement but had discharging ear.

April 12. Mastoid operation by Dr. Leifeld, sinus and dura exposed; apparently improved; no temperature, no headache, until April 21, when patient had slight rigor (possible from ice cream). Next day ***pain behind left eye,*** which continued off and on. Discharge from ear profuse; no dizziness; temperature 100.2°-102° nauseated and vomiting.

April 26. Lumbar puncture; fluid clear, not under pressure; globulin slighly increased; cell count, 5; no organisms; blood culture, negative; O. E. veins full, no indication of injection of disc. Hearing greatly reduced but present in left ear.

April 27. *Second Operation.* Eradication of deep cells of petrous. Decided not to open his dura. At completion of the operation I was not entirely satisfied; "the boy probably had a serous meningitis, although the low cell count—only three—would signify otherwise."

Granulation tissue found in the mastoid wound. The deep mastoid cells behind the external semicircular canals also opened. There was a large amount of carious bone along the zygomatic process where the dura was freely exposed. The sinus was covered by a thick granulation. Made a small opening in the sinus; had bleeding, but not very free.

April 28. Vomited. Had some dizziness following the operation. "He does not look entirely right." He had a white cell count of 17,000. Blood culture negative." I am still of the opinion that he has an involvement of the temporosphenoidal lobe."

April 29. "Since the operation the boy has been slightly dizzy, and on changing his position the dizziness is increased." Has a nystagmus on looking toward the right(away from the lesion) "It is possible that during the operation there was some injury done to his labyrinth, although I think not." Today appears slightly dopey, but his mother says he always sleeps considerably when sick. Temperature 101°. "I am afraid he is developing serous meningitis. It may be this is due to the injury of his labyrinth, because the deep cells of the apex of the petrous portion of the temple undoubtedly were involved."

Second Lumbar Puncture. Fluid under great pressure; macroscopically clear; globulin slightly increased. Sugar, normal. Cell count 322.5 per cubic mm. Smear shows no organisms. Culture sterile after 24 hours. Differential, polynuclears 93%, lymphocytes 7%.

April 30. Temperature irregular—90.2°-102°. Appears much better; is not dizzy. Total deafness of left ear.

Vestibular Reactions Spontaneous; rotary nystagmus to right on looking straight ahead; more noticeable on looking in right outer corner. *Spontaneous* deviation of *right hand two inches to left.* Tendency for left hand to deviate to *right.*

Rotating to Right. No perceptible change in the spontaneous rotary nystagmus; (possibly stopped for three or four seconds). No dizziness or nausea. Right hand deviated to left (see Spontaneous Deviation). Left hand deviated to right (see Spontaneous Deviation).

Rotating to Left. Spontaneous rotary nystagmus markedly increased for about 15 seconds; then about the same amplitude as before rotation. Dizziness. No nausea. Right hand deviated to left (no increase over spontaneous deviation). Left hand deviated to right (see Spontaneous Deviation).

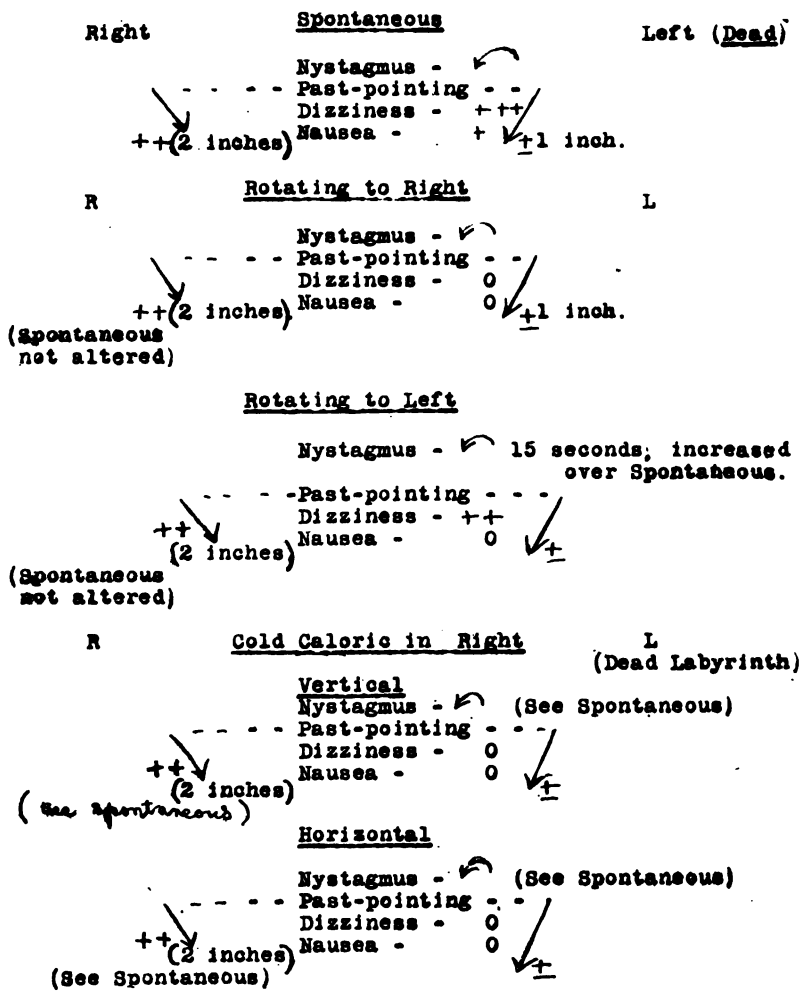
Cold Caloric in Right:

Head Erect: The spontaneous nystagmus to right was not altered; no change in spontaneous deviation; right deviated to left—left deviated to right; no dizziness; no nausea.

Head Back: No perceptible change in spontaneous nystagmus; right hand did not deviate (see Spontaneous Deviation); left hand deviated to right.

Chart I. G. C.

Graphic Representation of Above April 30:



May 2. Patient much better in every way, but still has irregular temperature—102°-102.2°. No dizziness. Leucocyte count, 13,400; differential, polynuclears, 78%; L. lymphocytes, 10%; S. lymphocytes, 11%; eosinophile, 1%.

Blood culture taken; negative. Spinal fluid; slight opacity of fluid, considerable pressure; globulin slightly increased. Sugar, normal. Cell count, 842 per cubic mm. No organisms in smear. Culture. Differential, polynuclears, 95%; lymphocytes, 5%.

May 5. Spinal fluid under considerable pressure; fluid opalescent and showed slight flocculent precipitate. Globulin slightly increased. Sugar normal. Cell count 600 per cubic mm. No organisms in smear. Fluid cultured; no organisms. Differential, polynuclears, 90%; lymphocytes, 10%.

May 6. Leucocyte count 8,800. Differential, polynuclears 72%; L. lymphocytes, 13%; S. lymphocytes, 14%; eosinophile, 1%. "This is the third lumbar puncture which has been done. At the first one before the operation for his labyrinth he did not have a high cell count. A short time after he had a cell count of 150. Three days later it rose to over 300. It is now over 600. The boy, however, looks perfectly well. He is pale. He has no other symptoms. In view of the fact that none of his labyrinthine symptoms came on until after the operation, and we know that he heard before that, and that his nystagmus developed the following day, and that he is dizzy, we had decided that it must be a protective process. We are impressed with this idea because of our experience with case J. G., Case VI, who after a labyrinthine operation, continued to have a high cell count with some rigidity of the neck. However, one month after the operation, he still had a cell count of 600. Some weeks later a small piece of sequestra from the labyrinth came away, and the man's condition immediately cleared. This makes us think that in a chronic case there is a protective meningitis going on along the posterior surface of the petrous portion of the temple. In opposition to this, however, is the fact that he had before the operation a severe pain behind his eye, that he had one chill, and was delirious. The diagnosis of the case must remain between three things; one, of protective meningitis, the result of the labyrinthine operation and the development of a protective area so as to cut off the ductus endolymphaticus, or the internal auditory meatus tract; second, an encapsulated abscess of the temporosphenoidal lobe, now at least of from four to six weeks' duration; or third an encapsulated serous meningitis, either along the posterior portion of the petrous or over the tegmen."

Vestibular Reactions:

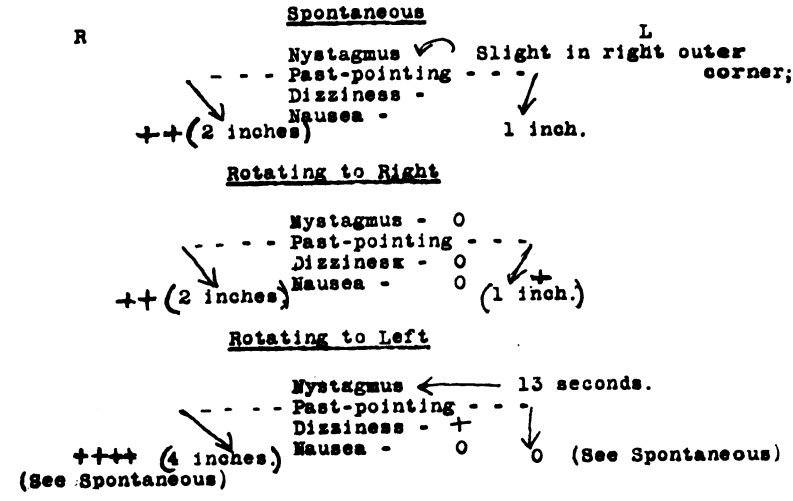
Head Erect: Spontaneous, slight nystagmus with rotary tendency to right in right outer corner. Spontaneous deviation of right, two inches to left.

Rotating to Right. No perceptible nystagmus; no dizziness or nausea. Left hand deviated to right; right hand deviated to left. (See Spontaneous Deviation).

Rotating to Left. Has nystagmus to right, of good amplitude, lasting 13 seconds. Slight dizziness; no nausea. Right hand deviated four inches to left; left hand did not deviate. (See Spontaneous Deviation).

Chart III. G. C.

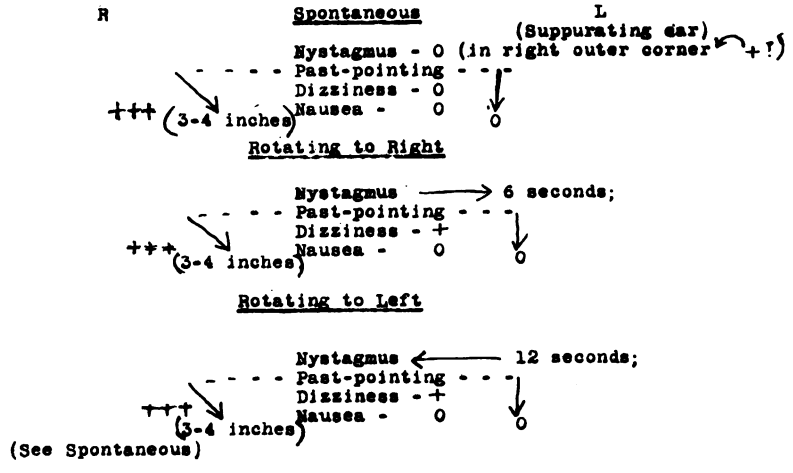
Graphic Representation of the Above:



May 7, 1921. Totally deaf. Slight nystagmus in looking to right; no adiadokokinesis; no heminanopsia of color on rough test; no pointing deviation; O. E. about same; veins full but not tortuous.

Chart III. G. C.

Wednesday, June 29, 1922.



Hearing:

	Right ear:	Left ear:
Watch -	Six feet	Totally deaf.
Whisper -	Across room	
Voice -	Across room.	

It would seem from studying the foregoing four cases that immediately on the increase of the intracranial pressure in protective meningitis or intrapiarachnoid abscess of the anterior surface of the cerebellum, (1) the nystagmus normally induced from the remaining functioning end-organ is reduced from the normal sixteen seconds to about half that duration—namely, to eight or nine seconds—and (2) the spontaneous past-pointing of the arm caused by the involvement of the cerebellar cortex situated posterior to the bony labyrinth cannot be altered by stimulation. Both of these observations would suggest a localizing diagnosis of the lesion.

A study of these cases furnished me a clue to that larger class of cases now so frequently seen, which, following an injury, such as fracture of the skull with cerebral trauma, or associated with a chronic progressive deafness, or from intoxications of all kinds, syphilis and so on; in fact, any condition which lessens the reactibility of the horizontal fibres to a particular form of stimuli (repeated turnings or repeated aerial flights, the clinical signs of which rapidly disappear)—immediately develop a great reduction in the duration of the nystagmus on turning; showing that while the fibres of the vertical canals apparently are much earlier attacked by an increase of the intracranial pressure when stimulated by the cold caloric, the horizontal fibres also are easily affected by any disturbance, as far at least as stimulation by turning is concerned.

Pointing Deviation:

B (Case IV) had bilateral past-pointing away from the lesion; homolateral—pointed inward; contralateral—pointed outward, with falling away from the lesion.

The spontaneous pointing deviation could not be altered by either turning or caloric. After opposite labyrinth became affected the homolateral arm of the second labyrinth was altered and pointed inward (away from the second lesion toward the first lesion), and again the spontaneous deviation could not be altered. In B the contralateral side apparently lost the capacity for stimulation. It stopped deviating in May, 1920; the contralateral continued reactible for a time.

C (Case VII) had bilateral past-pointing deviation, ***inward,*** which could not be altered. The contralateral side had a greater pointing deviation than the homolateral side. The homolateral side later could not be made to deviate at all, while the contralateral side still continued inward.

R (Case V). There was no past-pointing deviation, but the records are so few, and the child was so sick that it cannot be accepted as of any great value.

These four cases, in a general way, confirm the opinion that the spontaneous past-pointing deviation cannot be altered until at least the lesion has been removed as in the cases reported by Lowenstein. He, with Barany, thinks there is a spontaneous past-pointing deviation of the homolateral side outward.

The fact that B, C, and R could not have the spontaneous past-pointing deviations altered long after the inflammatory process had subsided shows that in these cases of protective meningitis the cerebellar cortex is affected (by the inflammatory process).

These cases would certainly tend to show that whatever mechanism is involved by turning, in diffuse suppurative disease in the meshes of the piarachnoid over the cerebellar cortex, this mechanism is suddenly impaired to such an extent that it does not recover. The influence of this past-pointing deviation would appear the same for both the spontaneous homolateral and contralateral—the past-pointing deviation could not be altered; and in some of the cases at least the homolateral deviation continued reactible while the contralateral was non-reactive.

II. *The Diagnostic Value of a Reduction of Intracranial Origin in the Duration of the Nystagmus from Turning in Diffuse Lesions of the Posterior Fossa:*

I am persuaded that in the cerebellar cortex of the posterior fossa there is something which has to do with this. It is probably in the cerebellar cortex because cortical lesions are the ones that induce it quickly (as in the cases reported), while in intra-cerebellar lesions such as intra-cerebellar abscess, this reduction of the obtainable nystagmus from turning is not present.

My own experience with this reduction in cases of fracture of the skull of the posterior fossa, leads me to believe that it is the filling of the piarachnoid pathways of the posterior fossa with either exudate (as in the herein reported cases of protective meningitis) or with blood as in linear fractures of the skull, chiefly of the posterior fossa, that gives rise to inflammatory and reactive changes which affect the cerebellar cortex; for the reduction of the duration of the obtainable nystagmus by turning occurs immediately, as proven in a case of meningitis which recovered

after subarachnoid irrigation. The reduction in the deviation of the nystagmus was demonstrated one week after the onset of the disease. When, however, the exudate is chiefly confined to the cerebrum, the reduction of the induced nystagmus from turning either does not occur at all or is not permanent as another case of suppurative meningitis which recovered following cerebral and spinal subarachnoid irrigation, had a normal deviation of the nystagmus a few months later.*

Induced Nystagmus from Turning and from Heat or Cold Physiologically and Diagnostically Different:

From a misconception we have in the past regarded the induced nystagmus from turning and from caloric as identical. This is physiologically and clinically incorrect, as I have herein demonstrated. There is no evidence to substantiate the view that the induced nystagmus from turning and from cold and heat are identical. We have presumed, however, that they were identical, because objectively they are apparently the same; but judging from physiological analogy it is much more likely that they are in certain parts, at least, separate. The form of stimuli are so dissimiliar—turning being a mechanical stimulus which is well adapted to cause a molecular disturbance in a fluid medium (such as the fluid of the labyrinth) analogous to a certain degree to stimulation of the proprioceptive sensory system —deep sensation— while cold and heat are thermic stimuli ill adapted to such disturbance, but, on the other hand, well adapted to stimulation of the nerve fibres by cutaneous sensation. As both are forms of sensory stimuli, the vestibular apparatus being one of the early specialized apparati to convert sensory impulse into voluntary and involuntary muscular contractions, the different sensory impulses from turning and caloric probably are transmitted by the vestibular apparatus in a similar way as the transmission of cutaneous impulses to the cord from pressure or thermal sensations, either by a different set of cutaneous nerves, or at least by nerves differentiated in the posterior roots.

It is possible that the cerebellar cortex *as a whole* has an analogous function to the posterior roots as far as the differentiaton of the vestibular stimulation or impulses is concerned. In turning we are stimulating in a way a muscular sense—the sense of movement, while in caloric we are stimulating a sensory sense—a cutaneous (thermic) sense.

*Eagleton, W. P. Subarachnoid Irrigation for Suppurative Meningitis. Trans. Amer. Otol. Soc., 1921.

TWO CASES ILLUSTRATIVE OF THE IMMEDIATE REDUCTION OF THE INDUCIBLE NYSTAGMUS FROM STIMULATING THE HORIZONTAL SEMICIRCULAR CANALS BY TURNING WITH PRESERVATION OF NORMAL HEARING IN DIFFUSE TRAUMATIC LESIONS OF THE POSTERIOR FOSSA, SUCH AS FROM LINEAR FRACTURE OF THE SKULL.

Case VIII. M. A., girl, 8 years of age.

Resume of Case:

Struck by an automobile; since then has had dizziness and headache, double vision in the distant, not in the near, which may not be pathological but looks abnormal; high degree of hypermetropia with astigmatism; reduced vestibular reaction in the horizontal canal; absence of reaction in vertical canal in left ear; hearing normal.

Impression:

"It would seem then that she undoubtedly had a fracture somewhere through into the posterior fossa. Both vertical and horizontal canals fail to function properly."

History:

In March, 1920, was run down by an automobile and badly injured, especially on the back of her head. She was taken to a drug store and about an hour afterward taken home in an automobile by a policeman. The mother does not know whether or not the child was unconscious at any time. The physician who was called in to attend her, and helped to take her home, attended her for a few weeks at her home. The back of the child's head was much swollen and discolored for more than a month, but the doctor did not regard it as due to a fracture of the skull. The child was in bed for a week after the accident. On getting up she had to be led, but the mother was unable to say whether this was due to dizziness, to general weakness, or to the injury to her right leg.

Patient seemed to recover gradually, but remained very pale, had no appetite, complained of pain beginning at the back of her head and coming over the eyes, and said she was invariably dizzy on first standing up. Since the accident she has been told at school that her sight is not normal. She does not see writing on the blackboard well, and her head aches after reading for a time. When asked if she ever sees double, she replied that "when things are far off they are two." A street car at a distance appears to be two cars, but becomes one as it draws near. The same is true of an automobile; also of persons; approaching at a distance she sees two, but when they are near by she sees them normally.

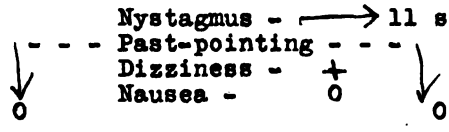
When she becomes excited or worried she feels a soreness or a stiffness in her neck and an aching in her left ear. She did not seem to have any ear symptoms at the time of the accident.

Jan. 11, 1921: Chart I. M.A.

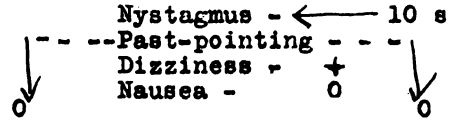
Vestibular Reactions;

No spontaneous nystagmus or past-pointing.

R. Rotating to Right: L



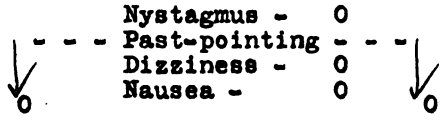
Rotating to Left



Cold Caloric in Left

R L

Vertical



Horizontal

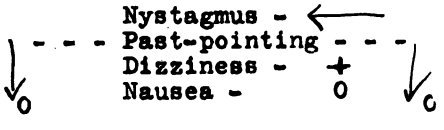
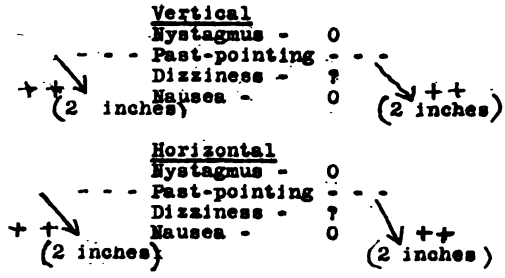


Chart II. M. A.

January 13:

R

Cold Caloric in Right



Feb. 16:

Report of Roentgen Examination:

"Plates were made of the head in both lateral positions and antero-posterior position with the occiput on the plate.

"They show a small line of fracture on the right side in the occipital bone just below the occipito-parietal suture. This is seen in both right lateral and antero-posterior views."

Chart III. M. A.

Feb. 18:

R L

Spontaneous

Nystagmus ⟶ Slight, in left outer corner.
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Right

Nystagmus - ⟶ Possibly 7 secs.; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Rotating to Left

Nystagmus ⟵ 7 or 8 secs.; slow movement;
Past-pointing - - -
Dizziness - 0
Nausea - 0

Cold Caloric in Right

R L

Vertical
Nystagmus -
Past-pointing - - -
Dizziness - ++
Nausea - 0

Horizontal
Nystagmus - ⟶
Past-pointing - - -
Dizziness - ++
Nausea - 0

Cold Caloric in Left

Vertical
Nystagmus -
Past-pointing - - -
Dizziness - + ?
Nausea - 0

Horizontal
Nystagmus - ⟵
Past-pointing - - -
Dizziness - +++
Nausea - 0

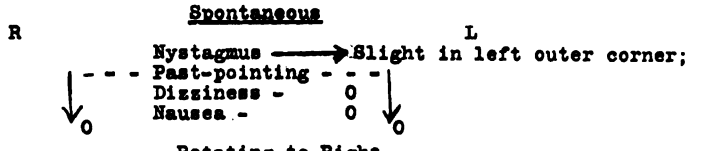
February 24:

Wasserman - negative.

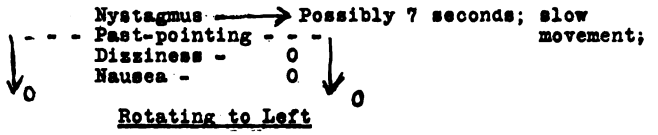
Chart IV M.A.

Feb. 29: **Vestibular Reactions**

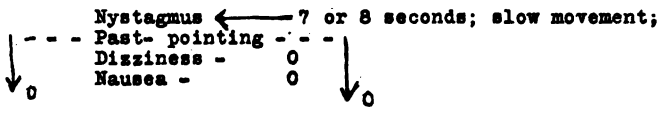
Spontaneous



Rotating to Right

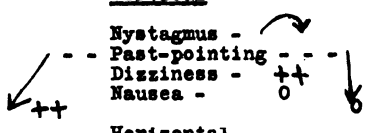


Rotating to Left

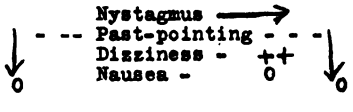


Cold Caloric in One Ear - the Right

Vertical



Horizontal



Case IX: J. N., fireman, 26 years of age.

History: Previous history of good health, with no history of acute illnesses Fell from third story window when infant of three years. Nov. 24, while driving engine which collided with trolley car, patient was thrown from engine to street. Was admitted to hospital suffering from abrasions, contusions and possible fracture of the skull.

Examination: General appearance of well-nourished, well-developed white male of 26, not acutely ill, lying quietly in bed, conscious and rational.

Head: Scalp, skull and hair, negative.

Eyes: Contusion over left eye, with a sub-conjunctival hemorrhage; pupils equal, regular, react to light, sclera clear in right eye, no nystagmus, no strabismus. Eye-grounds negative. No facial paralysis.

Neck, heart, lungs, abdomen, extremities, skin, all negative; nodes, not palpable.

Reflexes: Knee and ankle, active and equal; no Babinski; no clonus; no Oppenheim; abdominals and cremasteric active and equal. B. P. 140 over 90.

X-ray showed fracture right occipital region; no depression.

Provisional Diagnosis: (1) Contusion of left eye; (2) possible fracture of skull.

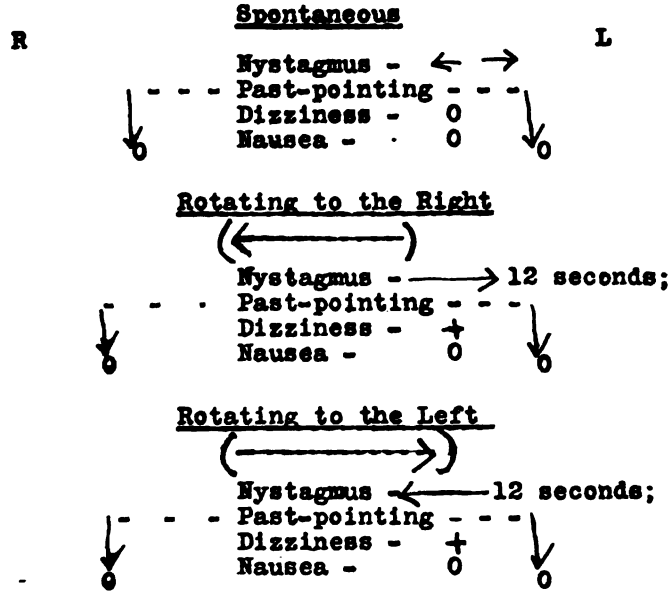
Remained in hospital from November 24 to December 3, when he was discharged.

After leaving hospital he complained of shooting pains from the top of his head to his neck, the pains lasting but a few minutes. Examination revealed dilated veins of the conjunctiva of the right eye, which were not noticed before. O. E. was negative.

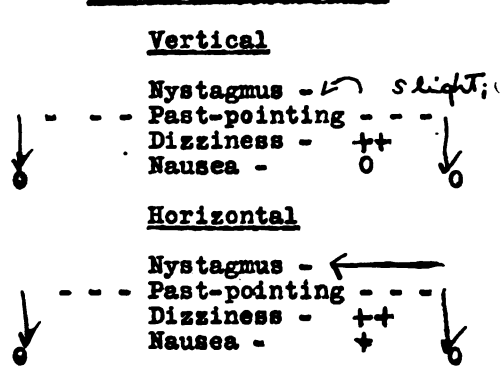
Vestibular Reactions.

Chart I. J.M.

December 31:



January 2:



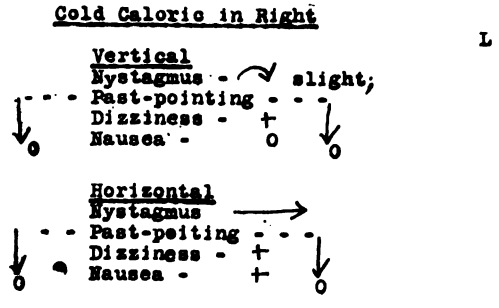
Note on Vestibular Reactions: Cold caloric in left ear gave a nystagmus but no pointing deviation. It seemed impossible to cause a pointing deviation in either arm. Both of the vestibular reactions were very much reduced.

The fact that the reduction on turning is of central, not peripheral, origin, is shown by the observation of Young,* who found that in cases of hereditary deaf-mutism the duration of the nystagmus on turning was not reduced, while in cases of deaf-mutism from a central disease, such as follows cerebro-spinal meningitis, it was not possible to cause an induced nystagmus on turning. Syphilitic deaf mutes may have a normal duration of the nystagmus, or no nystagmus at all, depending apparently upon the site of the lesion, whether peripheral or central.

That the reduction of the duration of the obtainable nystagmus

Chart II. J.M.

February 10:



may be due to intracranial involvement as well as readjusted compensation, and that it may occur immediately upon invasion of the meshes of the subarachnoid spaces by exudate or inflammation are shown by a case of protective or tubercular meningitis, referred to previously, treated by subarachnoid irrigation, rotated on the fifth day after the cerebro-occipito-allantoid irrigation and within a week after the appearance of the meningeal symptoms. In this case, with normal hearing the induced nystagmus obtainable was reduced to 8 and 13 seconds.

*Young, Calvin: Vestibular Reaction in Deaf-mutism. Journal Laryngology and Otology, November, 1921, Vol. 36, No. 11, p. 524.

Griffith (Laryngoscope, January, 1920, p. 22, and March, 1920, p. 129), demonstrated experimentally what had been clinically proved by Dunlap (Jour. A. M. A., Vol. 73, No. 1, p. 54), that repeated turning reduced and finally abolished the induced nystagmus as well as the vertigo and past-pointing.

Robertson, C. M. (Laryngoscope 32-105, Feb. 22; reviewed in Int. M. Educ. & S. Jour., April, 1922, 8b-157) also found reduction of nystagmus on repeated flights and turnings.

It is possible that the reduction occurs only when the virulence of the infection is confined chiefly to the meshes of the subarachnoid spaces of the posterior fossa as in the cases herein reported; while in the case of P (Trans. Amer. Otological Society, 1921) the inflammation following the line of fracture was confined mainly to the subarachnoid spaces of the cerebrum. In this instance, the pus and bacteria found in the lumbar puncture may not have been uniformly diffused over the cerebellar cortex to the extent and degree as when associated with protective and hemorrhagic lesions of the posterior fossa itself.

Conclusions and Deductions:

(1) In cases of increased intracranial pressure there is a reduction of the reactibility of the vertical canals of the side opposite the lesion, as evinced by the cold caloric when applied in the upright position with the head tilted forward 30°;

(2) When the lesion is situated above the tentorium, however, the reactibility, although reduced or delayed, is generally attainable. In increased intracranial pressure of the *posterior fossa* the reduction amounts to a complete absence of reactibility of the vertical canal of the unaffected side;

(3) This vestibular manifestation of increased intracranial pressure—namely, reduction of and in the posterior fossa, absence of reactibility of the vertical canals of both ears to the cold caloric—is a general diagnostic symptom of importance but is of no localizing value. Its presence on the other hand, largely prevents further localization by the vestibular tests alone.

(4) A protective collection of abnormal cerebrospinal fluid with partial obliteration of the cerebrospinal fluid circulatory system pathways from labyrinthine involvement, either suppurative or traumatic, situated over that portion of the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid, and manifested by a protective meningitis—shown by a high cell count but without microorganisms in the lumbar puncture—has a definite vestibular symptom complex.

(5) This vestibular symptom complex is:

(a) Loss of reactibility of the vertical canal of the opposite side, as well as of the affected side, from increased intracranial pressure;

(b) Early reduction of the maximum duration of the induced nystagmus, from stimulation of the opposite side from turning;

(c) The duration of the nystagmus from turning being only about 50% of that obtainable after destruction of one labyrinth, the reduction, therefore, is of intracranial and not labyrinthine origin;

(d) Both vestibular manifestations appear sufficiently early to be of diagnostic value and continue for a considerable period, and in certain cases are permanent;

(e) The accompanying spontaneous past-pointing deviation, from the intracranial involvement, rapidly disappears, probably by the establishment of compensation;

(f) Clinical evidence of the earlier presence of a spontaneous past-pointing deviation can be elicited by turning, as after a considerable period of time—after several years in one of my cases—it is impossible to induce a deviation in the opposite direction;

(g) The permanence of the latter vestibular manifestation —permanent inability to induce past-pointing of an extremity in the opposite direction to a previous spontaneous past-pointing deviation—may account for much of the confusion in regard to pointing deviations in all intracranial lesions, both gross and microscopic; if further observation confirms the suggestion here offered that inflammatory and infiltrating lesions of central portions of the vestibular apparatus sufficient to be accompanied by spontaneous past-pointing deviations possibly are never wholly recovered from—as shown by the absence of induced past-pointing in the opposite direction—while those from pressure such as that caused by tumors, endothelioma, cysts, and extradural hemorrhages, may entirely disappear;

(h) In the presence of a suppurative or traumatic lesion of one labyrinth, absence of reactibility of the opposite vertical canal to cold caloric, as well as of the affected side (associated with a 50% reduction of the maximum attainable duration of the inducible nystagmus from stimulation of the unaffected horizontal canal by turning—with or without an associated spontaneous pointing deviation), is a symptom complex of intracranial involvement of diagnostic value and suggests a protective meningitis with localized serous effusion—situated on the anterior surface of the cerebellum behind the posterior surface of the petrous pyramid near the internal auditory meatus;

(6) As intrapiarachnoid abscess from labyrinthine suppuration frequently is located in this situation, it is suggested that possibly the same vestibular symptom complex may be present here also; in which case—although this suggestion is purely from analogy and as yet has no clinical evidence to support it—this vestibular symptom complex would be of localizing value in distinguishing intrapiarachnoid abscess from intracerebellar abscess.

(7) The induced nystagmus from turning and caloric are fundamentally different because of the difference in the nature of the stimulation;

(8) Consequently, in diseases of the nervous system they should be regarded independently of each other, as they are capable of furnishing separate localizing diagnostic information.

(9) In the cerebellar cortex, or in that portion of it which is covered or reached by the meshes of the piarachnoid, is located a part of the mechanism which has to do with the *duration* of the induced nystagmus by turning. Choking of these meshes by inflammatory exudate (as in protective meningitis from labyrinthitis or from hemorrhage complicating linear fracture of the skull) reduces the duration of the inducible nystagmus immediately by about 50% of the normally obtainable nystagmus from turning. This sign is of great general diagnostic and medico- legal value.

(10) This reduction of the duration of the nystagmus from turning may be a fatigue (non-sustained), a muscular phenomenon, which is now recognised to be a part of the cerebellar functions of asthenia and astasia.

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